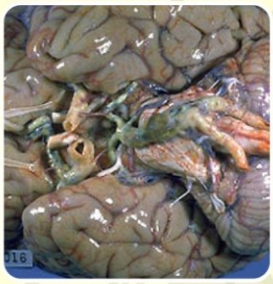
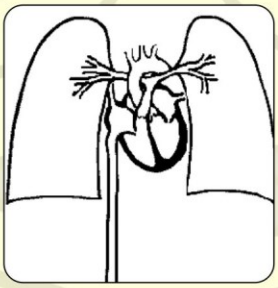
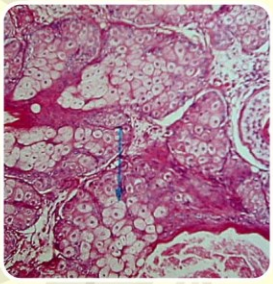
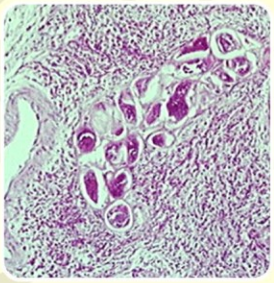
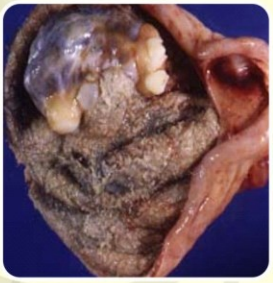
Academic board
CMDA Nigeria-Students
OAUTHC, Ile-Ife.

# Describe the lesion:
# Histopathology practicals

local publication

# PREFACE

There is a culture of eating meat last among us Nigerians for several reasons, chief among which is to encourage the eater to finish the meal (funny though). If morbid anatomy were a meal, the meat is to describe some lesion. This book can be likened to the meat in a Nigerian meal that should be eaten last.

Since morbid anatomy is such a unique field that apt description of lesions by students make them to shine like star before their examiner whether on paper or in person, this is an encouragement to the student that a quick revision note which links important stuffs read in patches up for the purpose of shinning in exams is available. It gives prototypical picture test questions with relevant answers, the way you're expected to answer them. ***Describe the lesion*** places emphasis on knowing how to pass morbid anatomy picture test well.

*The pathologist is interested not only in the recognition of structural alterations, but also in their significance"* - Stanley Robbins (1957).

So, this compendium is offered with the assumption that the reader must have done their homework well. It does not take the place of posting and standard texts that give a bird's eye view of structural alterations and their significance. Rather, it fulfills it purpose of being handy for quick perusal in preparation for test on pictures that have been used year in, year out in our great institution.

Permit me to place on record my sincere appreciation to Olasehinde Ifeoluwa Ayobami and his formidable team whose labor resulted in the birth of this piece, you're simply wonderful!

Go forth and shine!

M.B Nathaniel
School president 2016

# ACKNOWLEDGEMENT

Glory be to God for the grace He has given us to be able to start and finish this project. He alone deserves the glory.

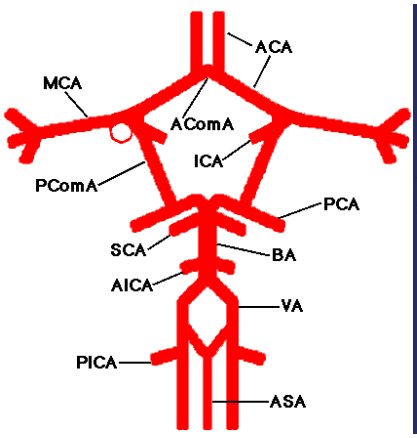
Appreciation to members of the academic board 2016 for their support through the period of writing this project. Special appreciation to the following people who have worked assiduously in making this project a reality- Jaja Erasmus, Oyeniyi Opeyemi, Ogunniran Damilola. Thank you so much. Surely, your labor of love isn't in vain.

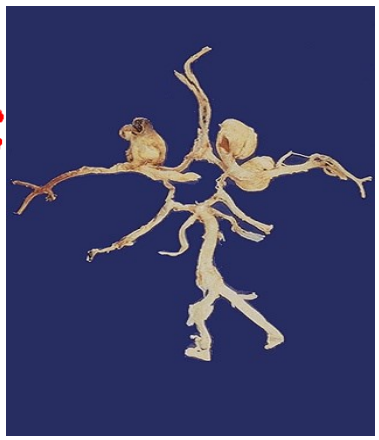
To our ever dynamic and amiable editor-in-chief, Akindele Toyosi, you're highly appreciated. God bless you.

To the entire central executive members for the year 2016, we say thank you for your support. God bless you.

OLASEHINDE IFEOLUWA A.

School Academic Secretary, 2016.



1 **Describe the lesion** – Slide shows a network of vessels supplying the brain with areas of abnormal saccular dilatations (called Berry aneurysm, a type of saccular aneurysm).

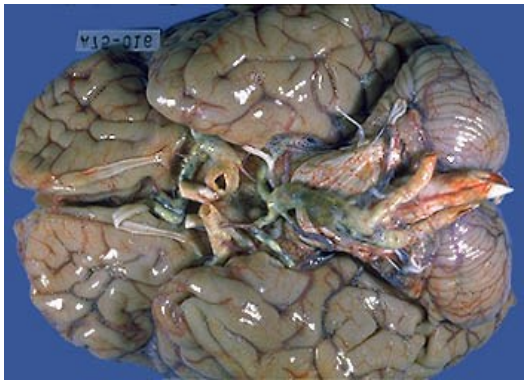
**Diagnosis** – Berry aneurysm

**Distribution** – They occur most commonly in the anterior circulation of the Circle of Willis. In descending order of frequency; between ACA & A comA – 40%, bifurcation of the MCA – 34%, between the ICA and MCA – 20%, bifurcation of Basilar artery –4%

**Associated disorders & Risk factors** – Coarctation of the aorta, Ehlers-Danlos (type IV), Neurofibromatosis (type1), Adult polycystic kidney disease, Marfan syndrome, Systemic hypertension, arteriovenous malformation, Fibromuscular dysplasia, cigarette smoking, Vitamin deficiency.

**Prognosis** – 1/3 die, 1/3 recover, 1/3 re-bleed

**Complications** – Subarachnoid hemorrhage, Stroke, Thrombus formation.



**2. Describe the lesion –** Slide shows the inferior surface of the brain with yellowish discoloration of the vessels of the circle of Willis due to accumulation of atheromatous plaque within the intima.

**Diagnosis** – Atheroma of the Circle of Willis

**Predisposing factors** –1. Non-modifiable e.g. Age (>40), Male gender, Family history, Genetics.

2. Modifiable e.g. Hyperlipidemia, hypertension, Smoking, Diabetes, Obesity, sedentary lifestyle, homocysteinaemia, Oral contraceptive use.

**Complications of the plaque** – Thrombosis, Hemorrhage, Rupture, Calcification, Ulceration, Aneurysmal formation, Embolism/Erosion.

**Clinical consequences** –1. Coronary vessels - Myocardial infarction, Coronary ischemic heart disease, Sudden cardiac death.

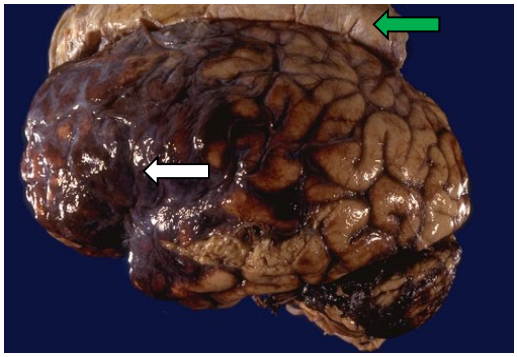
2. Circle of Willis – Cerebral infarction, cerebral hemorrhage, Chronic ischemia of the brain (multi-infarct dementia).

3. Renal vessel – Renal artery stenosis, chronic renal ischemia leading to hypertension, renal infarction.

4. Abdominal aorta – Gangrene, intermittent claudication.

5. Visceral arteries – Acute & chronic ischemia of bowel.

**3**



**Describe the lesion** – The slide shows the convex surface of the brain with areas of hemorrhage beneath the arachnoid mater. The reflected dura mater is seen to be devoid of blood stain.

**Diagnosis –** Subarachnoid hemorrhage

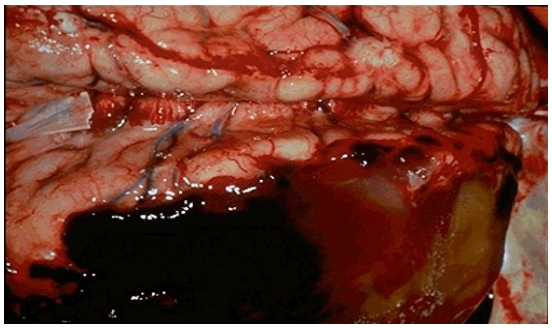
**White arrow** – Area of clotted blood

**Green arrow** – Reflected dura mater

**Causes** – Ruptured berry aneurysm, Hypertension, Trauma, Hemorrhagic disorders, arteriovenous malformation, intracerebral hemorrhage with secondary ventricular extension

**Clinical features –** Sudden (thunder-clap) headache, nuchal rigidity, neurological deficit, drowsiness, seizures

**4**



**Describe the lesion –** Slide shows the surface of the brain with areas of hemorrhage and blood-stained dura at the lower right corner.

**Diagnosis –** Subdural hemorrhage

**Cause –** Rupture of bridging veins

**Predisposing factors** – Trauma, Brain atrophy (in old age), bleeding disorders, Infants (weak vessels)

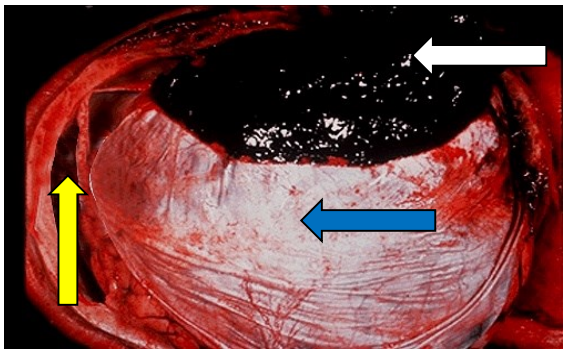
**Distribution** – Most common over the lateral aspect of the cerebral hemisphere. Only 10% are bilateral.

**NOTE: Differences between Subdural and subarachnoid hemorrhage**

| Parameters | SUBDURAL | SUBARACHNOID |
|---|---|---|
| Vessel involved | Veins | Arteries or veins |
| Appearance of dura | Blood-stained | Not blood-stained |
| Size of sulci | Not widened | Widened due to blood collection |
| *Water test | Positive | Negative |

*** Water test** refers to a crude method of differentiation in which water is poured on the surface of the brain. If the blood is washed off, it is a positive test i.e. subdural hemorrhage and if the blood persists, it is a negative test i.e. subarachnoid hemorrhage

**5.**



**Describe the lesion –**Slide shows a cut-open skull with the convex surface of the brain having areas of black brown hemorrhage on the dura mater.

**Diagnosis** – Epidural hemorrhage

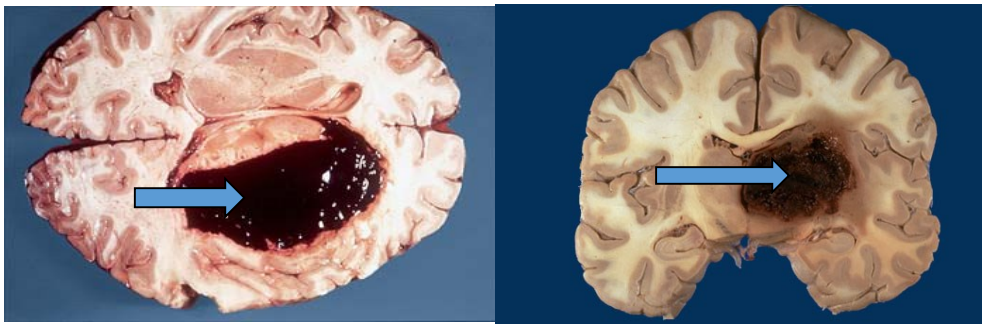
**Cause –** Trauma to the dural arteries, most commonly the middle meningeal artery

***This type hemorrhage is associated with Lucid interval before loss of consciousness (talk & die syndrome)**

**Complication -** Subfalcine cerebral herniation

**White arrow** – area of clotted blood
**Yellow arrow** - Frontal sinus
**Blue arrow** - Dura mater

**6.**



**Describe the lesion** – Cut surface of the brain focal area of hemorrhage within the region of the basal ganglia.

**Diagnosis** – Intracerebral hemorrhage

**Types**- Based on location, it could be lobar or ganglionic.

**Causes –** Hypertension (due to a ruptured Charcot-Bouchard aneurysm), Cerebral amyloid angiopathy, Infection, Vascular malformation, Bleeding diathesis and Primary brain tumors (Glioblastoma multiforme), Metastasis to the brain e.g. Renal Cell Carcinoma, choriocarcinoma.

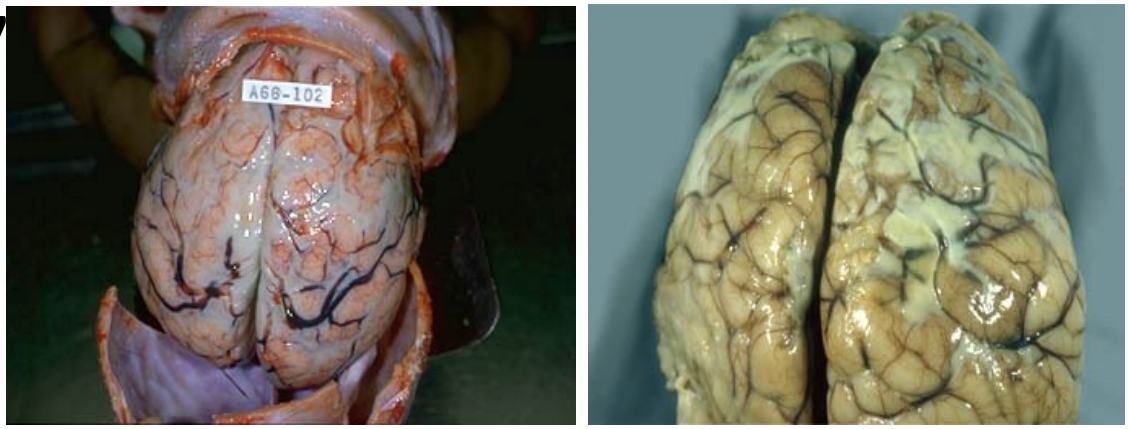
**Complications-** Cerebral edema, Compression of ipsilateral ventricle, Cerebral herniation

**Distribution-** In descending order; Basal ganglia, thalamus, pons and cerebellum

**Clinical features-** Severe headache, frequent nausea and vomiting, steady progression of symptoms over 15-20 minutes, coma.

**Blue Arrow-** Area of hemorrhage

**7**




**Describe the lesion -** Slide shows convex surface of the brain with areas of deposition of purulent material and distended vessels.

**Diagnosis -** Pyogenic meningitis.

**Complications -** Brain Abscess, Infarction, Cortical blindness, Cerebral palsy, Hemorrhage, Neurological deficit, hydrocephalus, leptomeningeal fibrosis, Waterhouse-Friderichsen syndrome, Encephalitis, DIC.

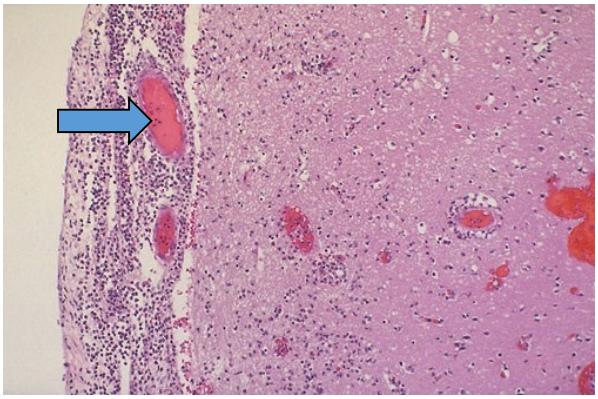
**Causative organisms**

| Age Group | Causes |
|---|---|
| Newborns | Group B *Streptococcus, Escherichia coli, Listeria monocytogenes* |
| Infants and Children | *Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenzae* type b |
| Adolescents and Young Adults | *Neisseria meningitidis, Streptococcus pneumoniae* |
| Older Adults | *Streptococcus pneumoniae, Neisseria meningitidis, Listeria monocytogenes* |

**Nonbacterial causes include –** Viral (Echovirus, Coxsackie), Parasitic (*Naegleria fowleri, Acanthamoeba spp.*)

**Findings;**

| CAUSE OF MENINGITIS | WBC COUNT | PRIMARY CELL TYPE | GLUCOSE | PROTEIN |
|---|---|---|---|---|
| Bacterial | Elevated | Neutrophil | Reduced | Elevated |
| Viral | Elevated | Lymphocyte | Normal | Elevated |
| Tuberculous | Elevated | Lymphocyte | Reduced | Elevated |
| Fungal | Elevated | Lymphocyte | Reduced | Elevated |

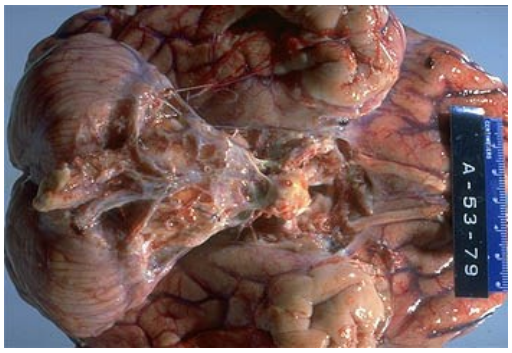
8. 

**Describe the lesion -** Histological slide showing brain tissue with massive inflammatory infiltrates with congested

blood vessels. There is edema and focal inflammation extending into the Virchow-Robin space in the cortex.

**Diagnosis** - Pyogenic meningitis

**Blue arrow** - shows engorged blood vessels.

**9.** 


**Describe the lesion -** Slide shows the basal surface of the brain with areas of massive suppurative exudates giving

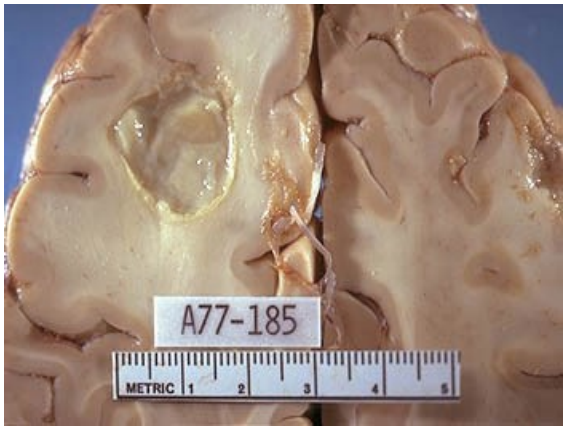
a spider-web appearance.

**Diagnosis** - Tuberculous Meningitis

**Complications -** Subarachnoid fibrosis causing non communicating hydrocephalus. Obliterating endarteritis causing infarction of the underlying brain.

**Findings -** refer to Number 7**.**

**Other causes of Basal Meningitis -** *Haemophilus influenza, Candida albicans, Cryptococcus neoformans*

**10.** 


**Describe the lesion -** Slide showing cut surface of the brain with a unilateral collection of purulent material or exudate within the substance of the brain tissue with a central area of necrosis surrounded by a thin wall.

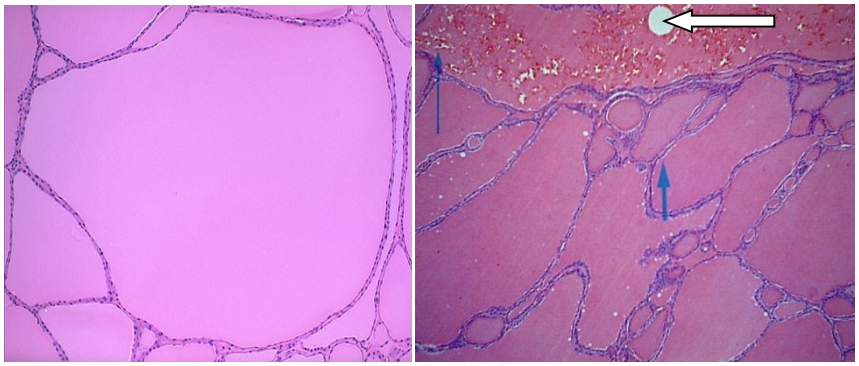
**Diagnosis -** Brain Abscess

**Causes -** Direct implantation of organism, hematogenous spread from primary site (heart, lungs), Local spread from contiguous structures (mastoiditis, paranasal sinusitis and poorly treated otitis media).

**Complication -** Seizures, Headache, Hemiparesis, Weakness of the contralateral limbs, impaired speech if speech areas are affected, raised intracranial pressure.

**Note** - Most common causative organism**s** in the immunocompromised patient are *Staphylococcus aureus* and *Streptococcus spp.*

**Diagnosis on CT Scan or MRI -** Ring enhancing lesion is seen.

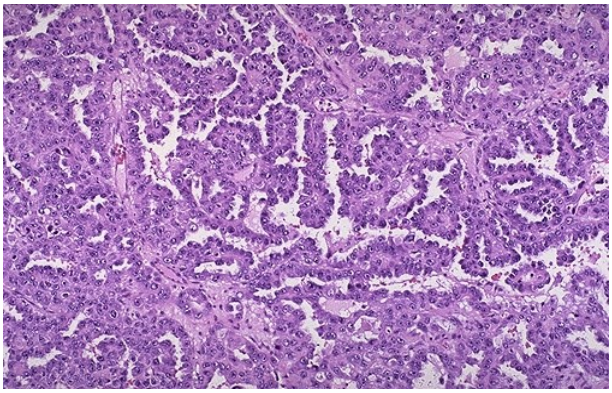
**11.** 

**Describe the lesion –** Slide shows abnormally and irregularly enlarge thyroid follicles with flattening of the lining epithelial cells and areas of hemorrhage (right image).

**Diagnosis** – Colloid goiter

**Gross appearance** – Enlargement of the entire gland without nodularity. It can progress to nodularity over time

**White arrow** – Cholesterol cleft
**Thin blue arrow** – Area of hemorrhage
**Thick blue arrow** – Flattened epithelium
**Risk factors** – Female gender, Iodine deficiency, Ingestion of goitrogens, Dyshormonogenesis

**12** 

**Describe the lesion –** Slide shows the section of a thyroid tissue with proliferation of malignant epithelial cells with hyperchromatic nuclei around a central fibrovascular core giving a finger-like pattern. Tumor cells have characteristic ground glass (Orphan annie) nuclei. Occasionally psammoma bodies are present.

**NOTE:** Other tumors where psammoma bodies are found include, Papillary renal cell carcinoma, papillary serous cystadenocarcinoma of the ovary, meningioma, ependymoma, mesothelioma, craniopharyngioma etc.

**Diagnosis –** Papillary carcinoma of the thyroid

**Variants** – Follicular, medullary, anaplastic and poorly differentiated (Insular) thyroid carcinomas

**Risk factors** – Female gender, radiotherapy

**13**

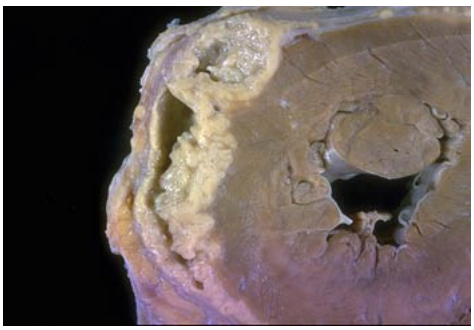
**Describe the lesion** – Slide shows the surface of the heart with deposition of fibrinous exudative material giving the characteristic 'bread and butter' appearance.

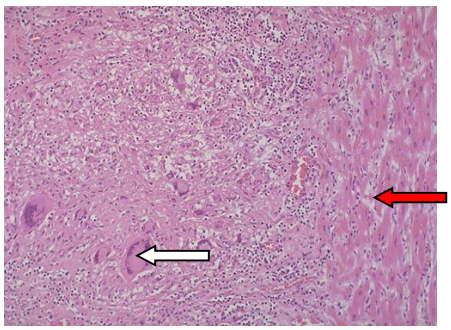
**Diagnosis** – Pericarditis

**Causes** – Infections (viruses, tuberculosis, bacteria, fungi, etc.), immunological (Rheumatic fever, SLE, Drug hypersensitivity reaction, Dressler syndrome), Others (uremia, Myocardial infarction)

**Complications** – Arrhythmia, friction rub, heart murmurs

**14**





**Describe the lesion –** Slide shows the cut surface of the heart with thickening of the myocardium and a well circumscribed lesion with central area of caseous necrosis.

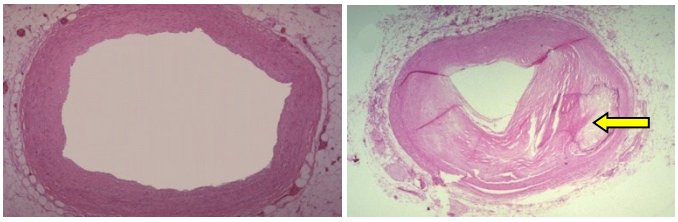
**Microscopy –** Slide shows section of the myocardial tissue with area of caseation and infiltrating inflammatory cells with presence of cells with horse-shoe shaped nuclei (Langhans cells)

**Diagnosis** – Tuberculous pericarditis

**White arrow** – Langhan cell
**Red arrow** – Normal myocardial tissue

**Complication** – Constrictive pericarditis

**15**



**Describe the lesion –** Slide shows transverse section of the coronary artery with reduced luminal diameter and thickened wall due to intimal deposition of atheromatous plaques. Slide on the left shows normal coronary artery

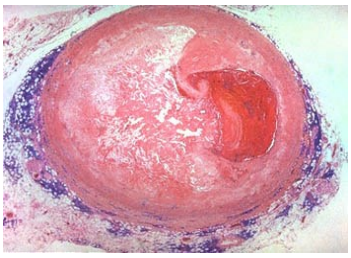
**Diagnosis –** Coronary atherosclerosis

**Stages –** 1. Initial lesion
2. Fatty streaks
3. intermediate lesion
4. Atheroma
5. Fibroatheroma
6. Complicated lesion

**Yellow arrow** – Atheromatous plaque

**Predilection of arteries for atherosclerosis** - Abdominal aorta, coronary arteries, popliteal arteries, thoracic aorta, Internal carotid artery, circle of Willis

**Complications –** Refer to 2

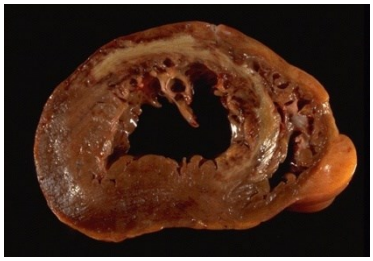
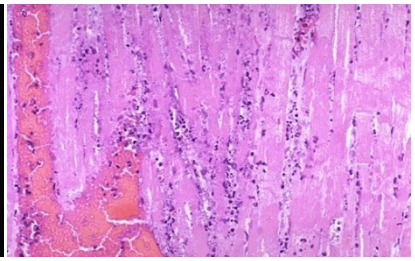
**16** 

**Describe the lesion –** Slide shows the transverse section of the coronary artery with a completely occluded lumen by a thrombus

**Diagnosis** – Coronary thrombosis

**Predisposing factors –** Virchow's triad

**Predilection of coronary arteries** – left anterior descending artery (45%), RCA (35%), LCA (15%)

**17**  

**Describe the lesion –** Slide shows the cut surface of the heart with a greyish white transmural area of infarction extending from the anterior ventricular wall into the interventricular septum.

**Microscopy –** Section myocardial tissue with loss of nuclei, areas of hemorrhage and inflammatory infiltrates and cellular debris

**Diagnosis** – Myocardial infarction

**Clinical features** – Chest pain, dyspnea, palpitations, diaphoresis

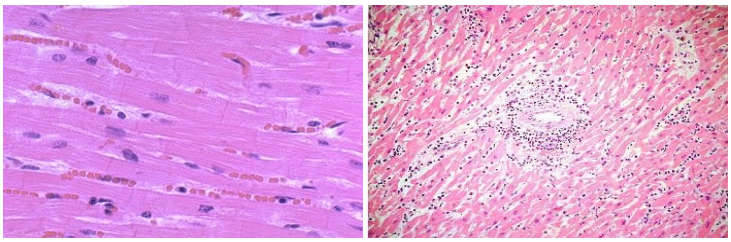
**Special stain used** – Triphenyl tetrazolium chloride stain

**Causes** - Coronary atherosclerosis, coronary artery spasm (Prinzmetal angina), Embolus

**Complications** – Arrhythmias, mural thrombosis, Cardiac failure, cardiac tamponade, cardiac aneurysm, Dressler's syndrome, Cardiogenic shock, cardiac rupture (4-7days Post MI).

**ECG findings** – ST segment elevation

**18**



**Describe the lesion –** Slide shows section of the myocardial tissue with perivascular inflammatory infiltrates. The nuclei maintain their normal position. The left slide showsnormal myocardium

**Diagnosis** – Myocarditis

**Causes**:

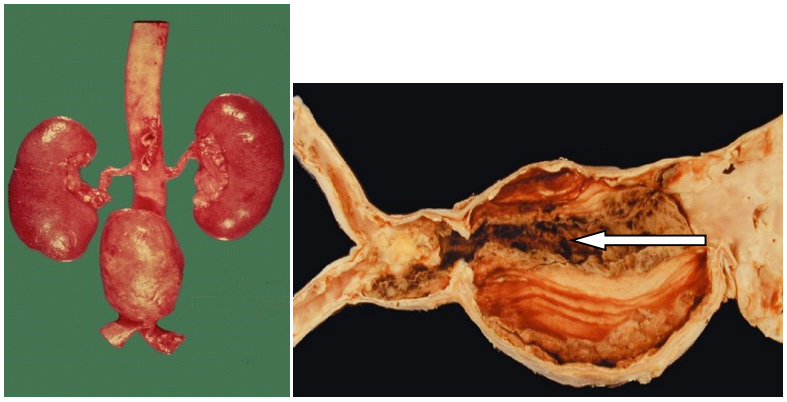
| Infections |
|---|
| Viruses (e.g., coxsackievirus, ECHO, influenza, HIV, cytomegalovirus) |
| Chlamydiae (e.g., *Chlamydophyla psittaci*) |
| Rickettsiae (e.g., *Rickettsia typhi*, typhus fever) |
| Bacteria (e.g., *Corynebacterium diphtheriae, Neisseria meningococcus, Borrelia* (Lyme disease) |
| Fungi (e.g., *Candida*) |
| Protozoa (e.g., *Trypanosoma cruzi* [Chagas disease], toxoplasmosis) |
| Helminths (e.g., trichinosis) |
| **Immune-Mediated Reactions** |
| Postviral |
| Poststreptococcal (rheumatic fever) |
| Systemic lupus erythematosus |
| Drug hypersensitivity (e.g., methyldopa, sulfonamides) |
| Transplant rejection |
| **Unknown** |
| Sarcoidosis |
| Giant cell myocarditis |

**19.** 

**Describe the lesion** – Slide shows three different aortas demonstrating mild, moderate and severe atherosclerosis from bottom to top. The aorta at the top shows extensive ulcerations in the plaques.

**Diagnosis** – Atheroma of the aorta

**Predisposing factors, Complications and clinical consequences** – Refer to 2

**20.** 

**Describe the lesion** – Left slide shows saccular dilatation of the aorta just above the birfurcation of the aorta and below the renal vessels. The right slide shows the cut section of the aorta with aneurysmal dilatation from a complicated atheromatous plaque with abundant layered mural thrombus within the aneurysm

**Diagnosis** – Abdominal aortic aneurym (AAA)

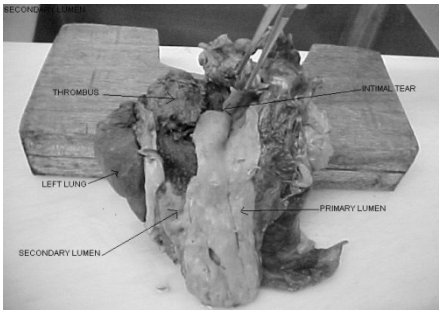
**Causes** – Hypertension, Atherosclerosis, Trauma, Vasculitis, Infections(mycotic aneurysm), Marfan syndrome, Ehlers-Danlos syndrome, Fibromuscular dysplasia

**Variants** – Inflammatory AAA, Immunoglobulin G4 related disease, mycotic AA

**Clinical features** – Asymptomatic, Rupture intoperitoneal cavity, Obstruction of a vessel branching offof the aorta resulting in ishaemic injury to the supplied tissue, embolism from atheroma or mural thrombus, Impingement on an adjaent tissue.

**White arrow** – Atherosclerotic plaque

**21.**




**Describe the lesion** – Slide shows the aorta with a longitudinal intra-luminal tear in the wall creating a secondary lumen in the aorta ('double-barrel aorta')

**Diagnosis** – Dissecting aortic aneurysm

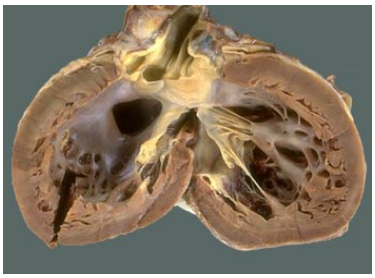
**Microscopic description** –There is cystic medial degeneration with an intramural hematoma

**Clinical features** – Severe tearing chest pain often radiating to the back between the scapula moving downward as the dissection progresses, rupture to pericardial, pleural and peritoneal cavities.

**Complications** – Cardiac tamponade, Aortic insufficiency, Myocardial infarction, Transverse myelitis

**Classification** – Type A (proximal – involving both ascending and descending aorta or just ascending alone), Type B (distal to the subclavian artery)

**22.**

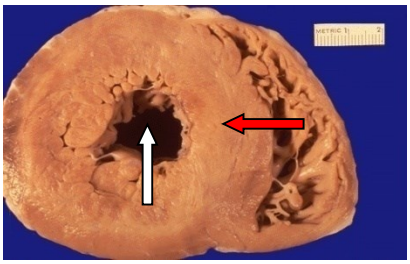


**Describe the lesion** – Slide shows the longitudinal section of the heart with a large defect in the membranous portion of the interventricular septum.

**Diagnosis** – Ventricular septal defect

**Note**: It is an acyanotic heart disease. Others include Atrial septal defect, Patent ductus arteriosus

**Complications** – Right ventricular hypertrophy, pulmonary hypertension

**23.** 

**Describe the lesion** – Slide shows the transverse section of the heart with concentric hypertrophy with a narrowed lumen.

**Diagnosis** – Concentric left ventricular hypertrophy

**Red arrow** – Left ventricular septal wall
**White arrow** – Left ventricular chamber

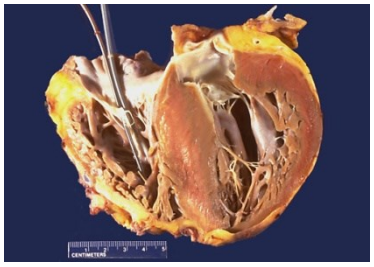
**Causes** – Systemic hypertension, Hypertrophic cardiomyopathy, Aortic stenosis

**Clinical features** – Cough, Hemoptysis, Dyspnea, Paroxysmal nocturnal dyspnea

**24.** 

**Describe the lesion** – Slide shows a longitudinal section of the heart with left ventricular hypertrophy and a small sized spleen (Cricket-ball spleen) beside it.

**Diagnosis** – Congestive cardiac failure secondary to left venticular hypertrophy

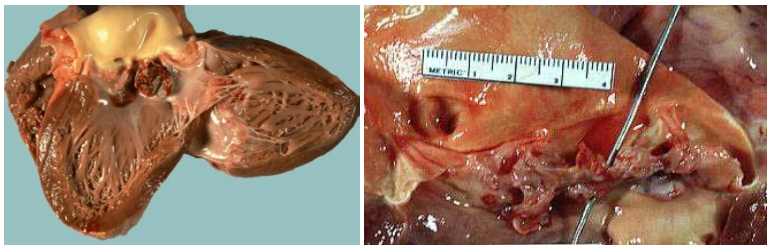
**25.** 

**Describe the lesion** – Slide shows a longitudinal section of the heart with marked left ventricular hypertrophy and asymetric bulging of the interventricular septum intothe left ventricular chamber giving a 'banana-like' configuration.

**Diagnosis** – Asymetric septal hypertrophy

*** **Causes** – Hypertrophic cardiomyopathy

**Microscopic description** – Massive myocye hypertrophy with tranverse myocyte diameter >40µm (normal = 15µm) with myofiber disarray and interstitial and replacement fibrosis

**26.** 

**Describe the lesion –** Slide shows large bulky friable vegetations on the line the line of closure of the heart valves eroding into the myocardium. On the right slide the aortic valve is perforated.

**Diagnosis –** Infective endocarditis

**Microscopic description** – It consists of 3 zones

- Outer layer consist of eosinophilic material composed of fibrin and platelets
- Basophilic zone – consisting of colonies of bacteria
- Deeper zone – consist of non-specific inflammatory reaction in the cusp of the valves

**Risk factors** – Rheumatic heart disease, mitral valve prolapse, bicuspid aortic valve, degenerative calcific aortic stenosis, congenital heart disease, artificial valves, indwelling catheters, dental procedures, immunosuppression, and intravenous drug use.

**Valves affected** – Mitral valve most commonly affected, followed by aortic valve, tricuspid valve and the pulmonary valve. ***Note*** that the tricuspid valve is the most commonly affected in Intravenous drug abusers

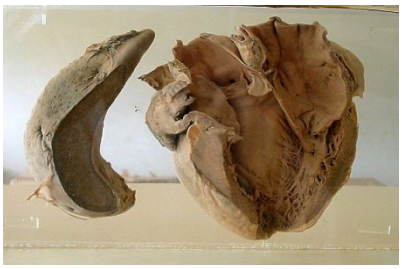
**Causative organisms** – In acute IE and IV drug users - *Staphylococcus aureus*

In subacute IE – *Streptococcus viridans*

In prosthetic valves - *Staphylococcus epidermidis*
Others include – Enterococci and HACEK group

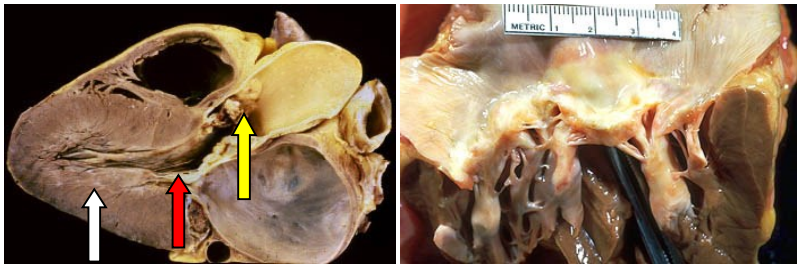
**Diagnostic criteria** – Duke's criteria (2 major or 1 major + 3 minor or 5 minor criteria)

**Complications** – Valvular perforation, Myocardial abcess formation,suppurative pericarditis, congestive cardiac failure.

**27.** 

**Describe the lesion** – slide shows the longitudinal section of the heart with small warty vegetations in the mitral valve with an enlarged spleen beside it.

**Diagnosis** – CCF secondary to Rheumatic heart disease

**28.** 


**Describe the lesion** – The left picture shows the longitudinal setion of the heart with small warty vegetation on the mitral and aortic valves. The right picture shows small multiple greyish-brown vegetations firmly attached along the line of closure of the mitral valve

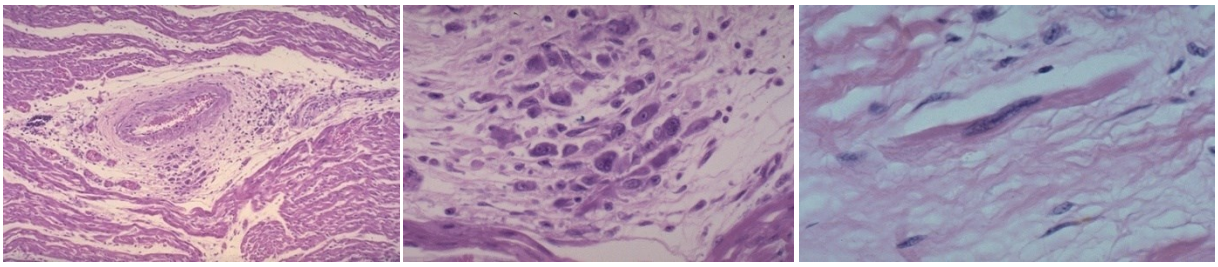
**Diagnosis** – Rheumatic heart disease

**White arrow** – Normal caridiac muscle in the left ventricular wall
**Red arrow** – Mitral valve
**Yellow arrow** – Aortic valve

**Causative organism** – Group A beta hemolytic *Streptococcus*

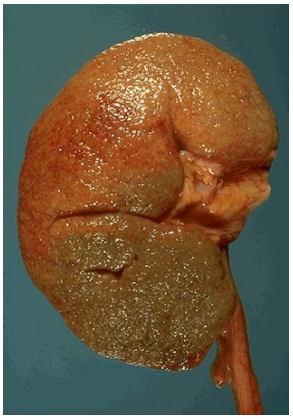
**Diagnostic criteria/Clinical features** – Jones' criteria (2 Major or 1 major + 2 minor criteria)

**Complications** – Mitral stenosis, Aortic stenosis, Congestive cardiac failure, Infective endocarditis

**29.** 

**Desscribe the lesion** – Slide shows an area of fibrinous necrosis surrounded by lymphocytes, plasma cells, anitshckow cells (left and middle images – Aschoff body). The right slide (anitshkow cell) shows unusual cells within the aschoff body whose nuclei contain a central band of chromatin (caterpillar cells)

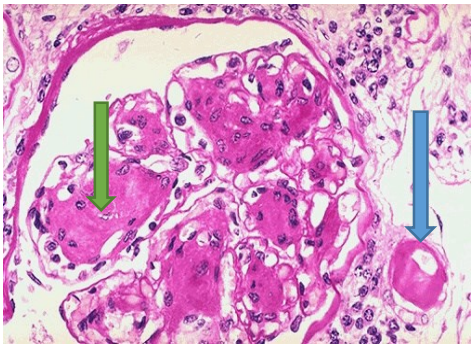
**Diagnosis** – Rheumatic heart disease

**30.** 

**Describe the lesion** – slide shows kidney with surface showing diffuse fine granularity that resembles grain leather.

**Diagnosis**: benign nephrosclerosis

**Conditions where it can be seen**: benign hypertension, Diabetes mellitus, aging.

**31.** 

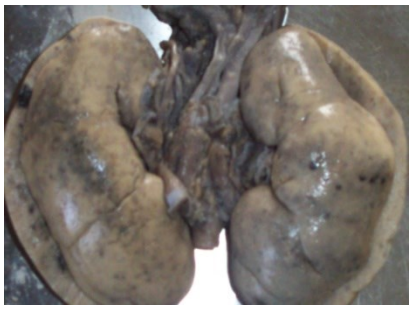
**Describe the lesion** – slide shows histological section of glomerulus with basement membrane thickening and increased mesangial matrix. There are ovoid,spherical hyaline masses in the glomerulus which eventually obliterates the glomerular thuft. At the lower right corner is an arteriole thickened with hyalinised material.

**Diagnosis**- nodular glomerulosclerosis showing hyaline arteriolosclerosis.

**Blue arrow**- thickened arteriole

**Green arrow**- hyaline mass in glonerular thuft.

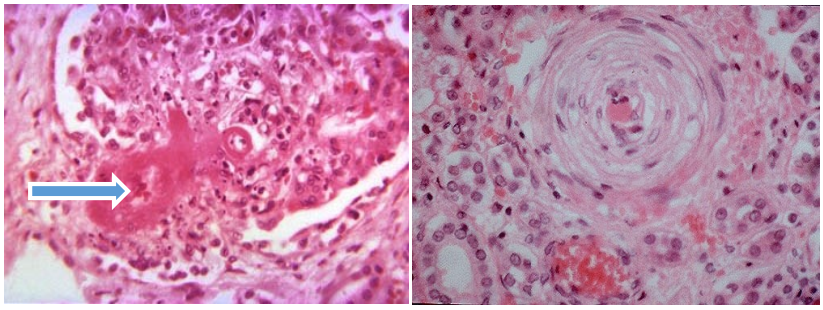
**Stain used**- periodic acid schiff stain.

**32.** 

**Describe the lesion** – slide shows surface of the kidney which smooth with subscapular petechial hemorrhages. This appearance is called the 'flea-bitten' appearance.

**Diagnosis** : malignant hypertension

**Clinical features**: papilloedema, convulsions, coma, nausea, vomitting, hematuria, albuminuria, hemolytic anemia.

**33.** 

**Describe the lesion – Left slide** shows smudgy eosinophilic appearance of the vessel wall due to fibrin deposition with a thrombosed glomerular vessel. The **right slide** shows a

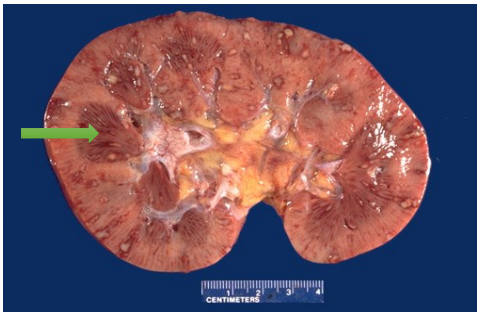
vessel exhibiting concentric laminated thickening of the vessel wall with luminal narrowing giving a characteristic 'onion-skin appearance'.

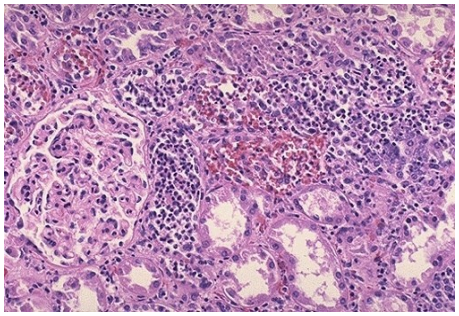
**Blue arrow** – Thrombosed vessel

**Diagnosis** – Malignant hypertension

**Clinical features** – SBP > 200mmHg, DBP > 120mmHg
Papilloedema
Retinal hemorrhages
Renal failure
Encephalopathy.

**34.**



**Describe the lesion** – The **left slide** shows the gross morphology of the cut section of the kidney with many small yellowish microabscesses in both the cortex and medulla. The **right slide** shows the microscopic section of the kidney with infiltrating polymorphonuclear cells seen in tubules .

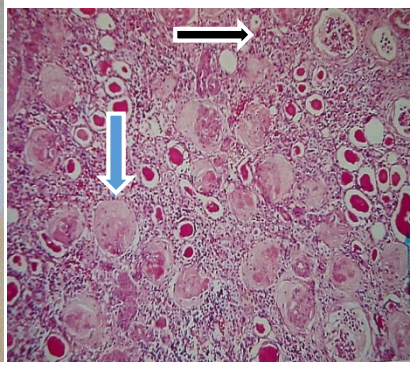
**Green arrow** – Renal pyramid

**Diagnosis** – Acute pyelonephritis

**Predisposing factors** – Urinary tract obstruction
Previous instrumentation
Diabetes mellitus
Pregnancy
Increasing age
Vesicoureteral reflux

**Complications** – Papillary necrosis, (*N.B; Other causes of papillary necrosis includes DM, Sickle cell disease, Analgesic nephropathy, Sepsis, Gastroenteritis, Urinary tract obstruction)*
Chronic pyelonephritis
Pyonephrosis
Pyonephritis
Perinephric abscess formation
Metastatic abscesses

**Causative organisms** – *E.coli* (most common), others include *Klesiella spp, Proteus spp, Enterobacter, Streptococcus faecalis*

**35.**  

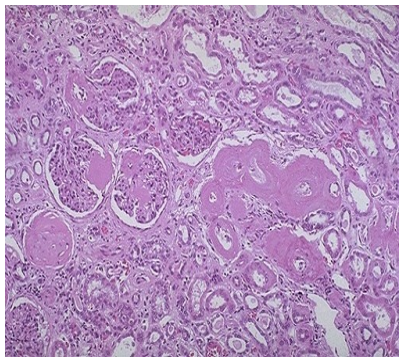
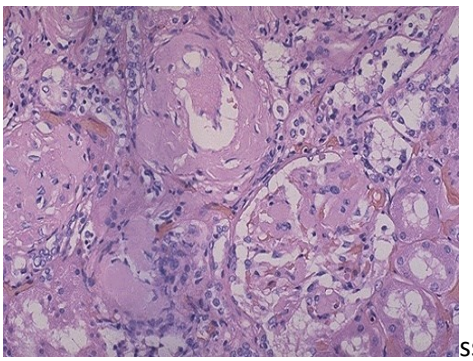
**Describe the lesion** – The **left slide** shows the gross morphology of the kidney that is shrunken with ill-depressed cortical scarring and deformity of the pelvi-calyseal system. The **right slide** shows the microscopic view of the kidney with tubular atrophy characterised by thyroidization (also seen in struma ovarii) of the renal tubules due presence eosinophilic materials, and chronic inflammatory cell infiltrates.

**Blue arrow** – Obsolescent glomerulus

**Black arrow** – Normal glomerulus

**Diagnosis** – Chronic pyelonephritis

**N.B***: Xanthogranulomatous pyelonephritis* is an uncommon variant of chronic pyelonephritis and is characterised by accumulation of foamy macrophages admixed with other inflammatory cells and giant cells. It is often associated with *Proteus* infection.

**36.**   ss

**Describe the lesion** – The slides show abnormal deposition of proteinecious hyaline material (amyloid) in the extracellular space of the kidney tubules including the glomeruli and the vessels.

**Macroscopic description-** The kidneys may be of normal size, enlarged or shrunken because of ischemia caused by vascular narrowing due to deposition of amyloid in arterial and arteriolar walls. Cut surface is pale, waxy and transluscent.

**Staining-** Congo red staining

**Diagnosis-** Amyloidosis of the kidney

**Forms of amyloid protein-** 1. Amyloid light chain- associated with certain froms of plasma cekll tumors

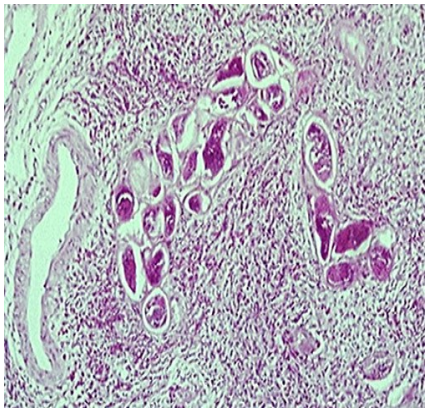
2. Amyloid associated protein- associated with chronic inflammation, often called secondary amyloidosis.

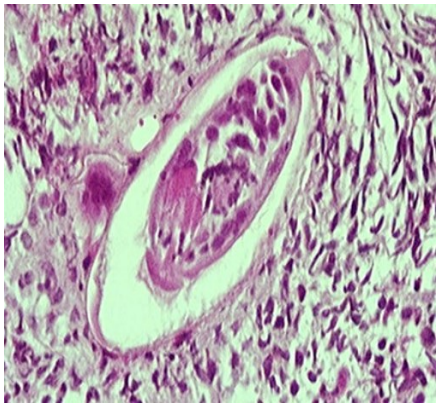
3. β amyloid protein- associated with Alzheimer disease

4. Transthyretin- found in the heart of aged individuals

5. β2 microglobulin- associated with patients on long term hemodialysis.

**37.**





**Describe the lesion** – The left slide show histological section of the bladder with eggs of *S. hematobium* in the bladder wall and adult worms in the vessels of the bladderand also plenty eosinophilic infiltration. The right slide shows egg of *S. hematobium* characterised by its terminal spine that differentiates it from the eggs of other Schistosoma species.

**Diagnosis**- Schistosomiasis of the bladder

**Infective Stage**- Cercaria

**Complications**- 1. Inflammation and fibrosis of the ureteral walls leading to ureteral obstruction, hydronephrosis and chronic pyelonephritis.

2. Increased risk for squamous cell carcinonma of the bladder.

**38.** 

**Describe the lesion** – slide shows cut surface of an enlarged kidney with a characteristic variegated appearance of soft fishflesh-like, greyish-white tumor with foci of necrosis and hemorrhage and grossly identifiable myxomatous elements.

**Microscopic desciption**- there is a triphasic mixture of blastemal, stromal and epithelial cell types.

**Diagnosis**- nephroblastema( Wilms tumor).

**Peak age**- 2-5 years. 95% cases occur before 10 years. It has equal sex incidence.

**Affected genes**- WT1 gene and WT2 gene, both on chromosome 11p.

**Associated syndromes**- Beckwith-widemann syndrome, WAGR syndrome, Dennys-Drash syndrome.

**Associated malignancies**- osteogenic sarcoma, retinoblastoma, sarcoma botryoides.

**Clinical features**- large abdominal mass, abdominal pain, total and painless hematuria(schistosomiasis is associated with terminal hematuria), hypertension.

**Prognosis**- excellent, with long term survival rate of 90%.

**39.** 

**Describe the lesion-** slide shows cut surface of the kidney which is enlarged and shows cysts varying in size, containing clear to brown fluid, throughout the renal parenchyma with the renal pelvis and calyces greatly distorted by the cysts and also loss of the cortico-medullary differentiation.

**Microscopic description-** cysts are lined with cuboidal or flattened epithelium and they may have papillary projections. Functional nephrons exist between the cysts with areas of interstitial fibrosis and chronic inflammation.

**Diagnosos-** adult polycystic kidney disease.

**Mode of inheritance-** autosomal dominant.

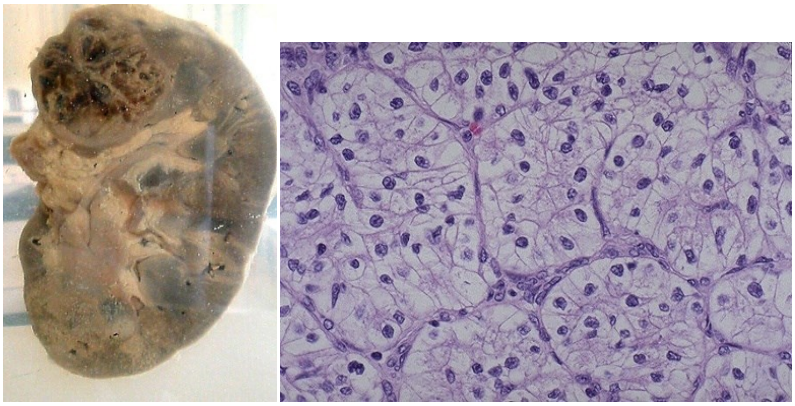
**Affected genes-** PKD1( codes for polycystin 1) on chromosome 16p and PKD2( codes for polycystin 2) on chromosome 4q.

**Clinical features-** 1. Usually asymptomatic until the 3rd or 4th decade of life.
2. renal insufficiency 3. Hematuria 4. Abominal pain
5. hypertension

**Extrarenal manifestations-** 1. Von Meyenburg complexes in the liver( 97%)
2. Hepatic cysts( 40-88%)
3. berry aneurisms(10-30%)
4. Mitral valve prolapse(20%)
5. intestinal diverticula.

**Cause of death:** 25% die from infection, 40% from hypertension and heart disease, 15% from ruptured berry aneurysm or stroke.

**40.**



**Describe the lesion** – the slide on the left shows the cut surface of a kidney. There's a well circumscribed and well differentiated tumor at the upper pole, golden yellow in color with a variegated appearance characteristic of the tumor and areas of necrosis and hemorrhage. The right slide shows a histologic section of the kidney showing tumor cells which are polygonal in shape and have clear and abundant cytoplasm which is vacuolated with indistinct cell borders.

**Diagnosis**- Renal cell carcinoma aka Grawitz tumor/hypernephroma/nephrocellular carcinoma.

**Histological subtypes**: 1. Clear cell carcinoma(70-80%) 2. Papillary carcinoma( 10-15%)
3. Chromophobe carcinoma(5%) 4. Sarcomatoid RCC (1.5%)
5. collecting duct(Bellini duct) carcinoma(0.5%)

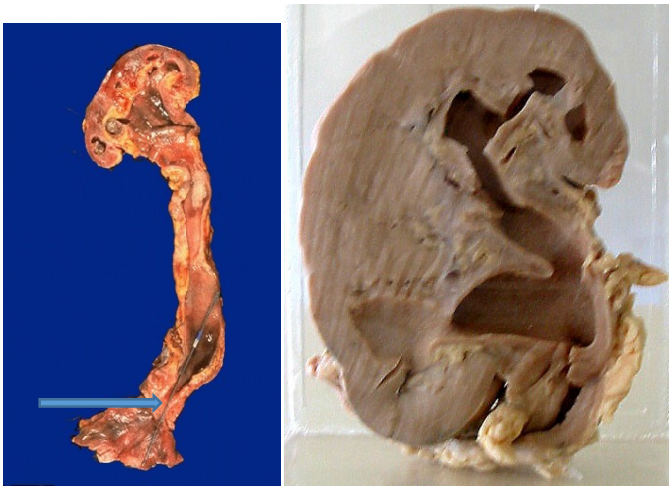
**Risk factors-** 1. cigarette Smoking 2. Genetic susceptibilty- von hippel-Lindau disea 3. Obesity

4. Exposure to asbestos 5. Acquired cystic disease 6. Chronic kideney disease

**Clinical features-** 1. Classic triad- costovertebral pain, palpable mass, hematuria( 10%) 2. Anemia 3. Weight loss 4. Paraneoplastic syndromes – cushing syndrome, hypercalcemia, hypertension, gynecomastia, leukemoid reaction, polycythemia, amyloidosis, Stauffer syndrome( hepatomegaly with hepatic dysfunction)

**Site of metastasis-** lungs( > 50%), bones( 33%), regional lymph nodes, liver, adrenal glands, brain.

**41.**

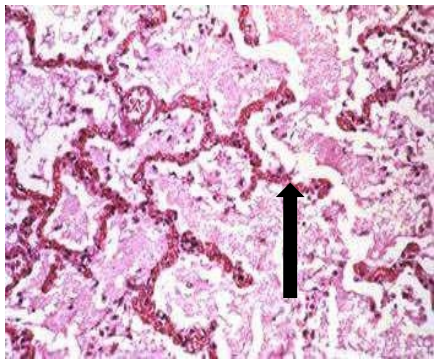
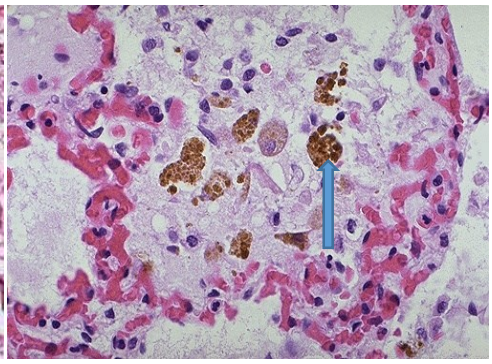


**Describe the lesion** – left slide shows upper urinary tract with enlarged ureter and kidney with reduced parenchyma. Right slide shows cut section of the kidney with cystic dilation of renal pelvis and calyces and associated atrophy of the kidney.

**Diagnosis**- hydroureter(left) and hydronephrosis(right).

**Causes**-bladder neck obstruction, posterior urethral valve, vesicoureteral reflux, pregnancy, benign prostatic hypertrophy, urehthral stricture.

**Blue arrow**- ureteral orifice.

**42.**  

**Describe the lesion** – slides show histological section of the alveoli filled with a smooth to slightly floccular pink material characteristic for pulmonary oedema with capillaries in the alveolar walls congested with many red blood cells. Hemosiderin-laden macrophages( heart-failure cells) are seen in the alveolar spaces of the lungs.

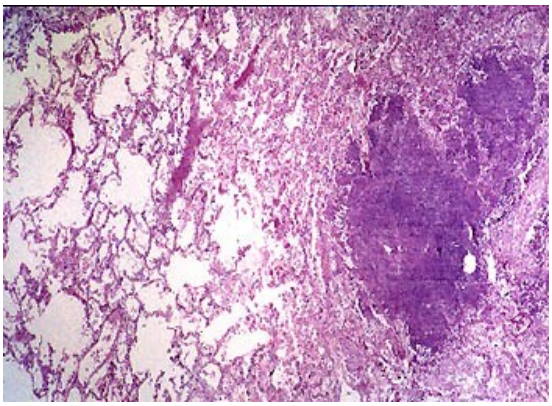
**Gross description**- lungs are firm, brown, heavy and wet. Cut sections shoe hemorrhagic areas due to abundant extravasation from alveolar capillaries.

**Diagnosis**- chronic venous congestion of the lungs.

**Causes**- left-sided heart failure( commonest), mitral venosis, fluid overload.

**Blue arrow**- hemosiderin-laden macrophages

**Black arrow**- engorged alveolar capillaries.

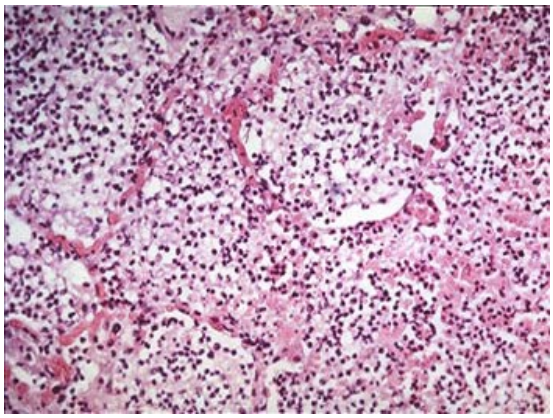
**43.**  

**Describe the lesion** – left slide cut section of the lung showing patch areas of consolidation centred on bronchioles across all the lobes interspersed with uninvolved areas. Right slide shows microscopic description with neutrophilic infiltration of the bronchioles and surrounding alveoli.

**Diagnosis**- Bronchopneumonia. It is usually seen in the extreme of ages and the immunosuppressed.

**Aetiology**- *Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae, Klebsiella pneumoniae, Pseudomonas aeruginosa.*

**Complications- 1.** carnification from organisation of exudate 2. Lung abscess
3. Empyema thoracis **4.** Pulmonary fibrosis 5. Septicemia
6. Metastatic abscess 7. Bronchietasis

**44.**  

**Describe the lesion** – left slide shows a lobe of the lung; there is consolidation of the entire lobe of the lung

Right slide shows microscopic description with intra-aveolar suppurative inflammatory infiltration of neutrophils and oedema.

**Diagnosis-** Lobar pneumonia. Associated with high virulence organsims and often seen in middle-aged.

**Four stages of lobar pneumonia evolution-** 1. Congestion 2. Red hepatisation 3. Grey hepatisation 4. Resolution

**Aetiology-** *Streptococcus* pneumoniae(95%), *Klebsiella pneumoniae*( seen in chronic alcoholics), *Haemophilus influenzae*.

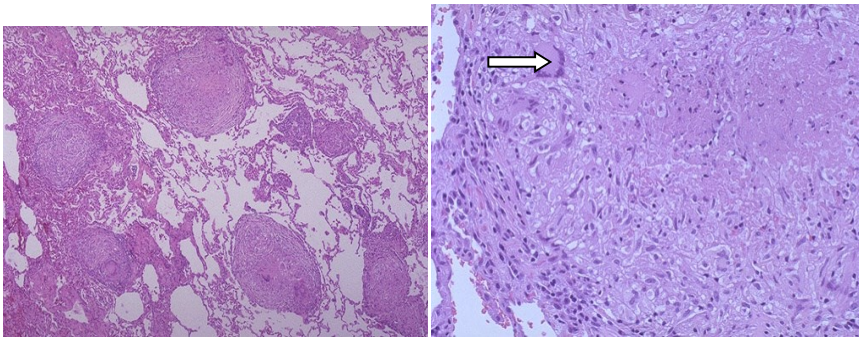
**Complications- 1.** Lung abscess 2. Empyema thoracis 3. Suppurative pericarditis 4. Septicemia **5.** Pyogenic meningitis 6. Peritonitis 7. Acute endocarditis

**45.**

**Describe the lesion** – left slide shows gross morphology of the lung with multiple granuloma and areas of caseous necrosis and also cavitatory lesions. Right slide shows gross morphology of the lung with general consolidation, areas of collapsed cavity, area of cavitations, dark nodular lesions of the hilar area( hilar lymph nodes).

**Diagnosis**- Pulmonary tuberculosis

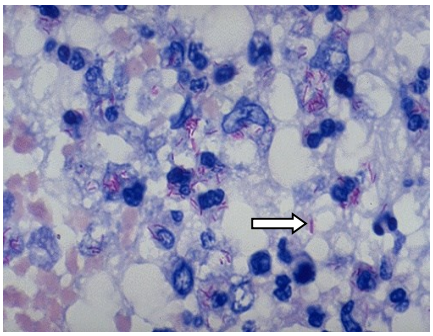
**46.**



**Describe the lesion** – Slide shows the lung tissue with multiple granulomas (left slide), Slides shows microscopic aggregations of inflammatory cells which are surrounding a central area of caseous necrosis with the presence of giant cells (Langhan cells).

**Diagnosis** - Pulmonary tuberculosis

**White arrow** – Langhan cell (giant cell)

**47.** 

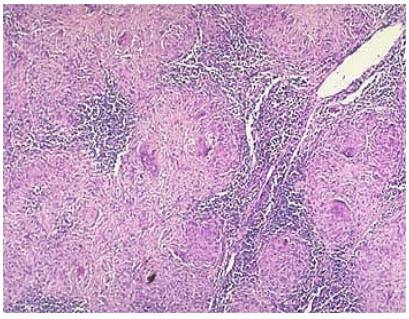
**Describe the lesion** – Slide shows a Ziehl-Neelson stained section showing bean-shaped macrophages(blue) with pinkish tubercle bacilli.

**Diagnosis** – Pulmonary tuberculosis

**White arrow** – Tubercle bacilli/Mycobacteria tuberculosis/Acid fast bacilli

**What media can be used** – Lowenstein-Jensen medium, BACTEC,

**Composition of the stain used** – Carbofuscin(primary stain), 3% HCl(decolourizer), malachite green/methlene blue(counterstain).

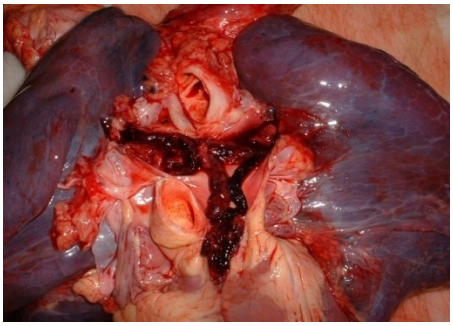
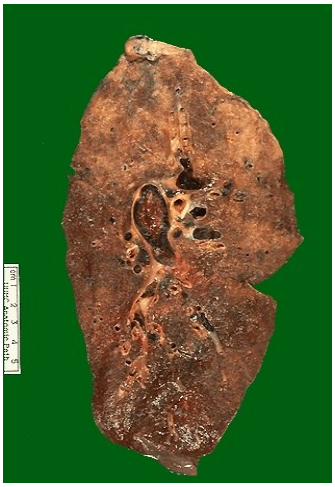
**48.** 

**Describe the lesion** – slide shows non-caseating epitheloid granulomas with tightly packed epithelioid cells, langhans giant cells and lymphocytes.

**Diagnosis**- sarcoidosis

**NB:** Schaumann bodies and asteroid bodies are seen in sarcoidosis

**Differential diagnosis**- atypical microbacteria, berrylliosis, extrinsic allergic alveolitis.

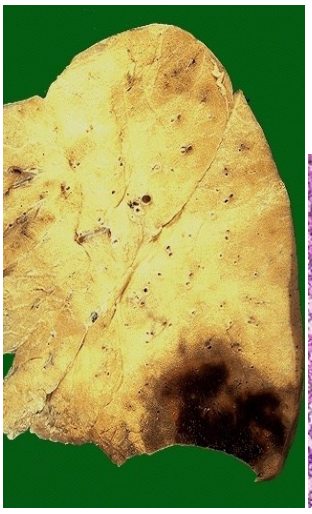
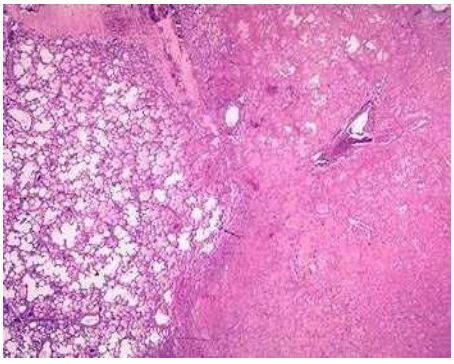
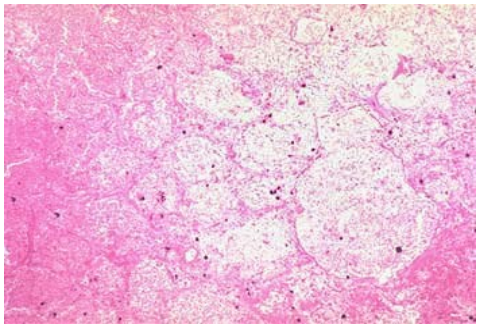
**49.**  

**Describe the lesion –** Slide shows the gross picture of the lungs with a clot(pulmonary embolus) in the pulmonary artery

**Diagnosis –** Pulmonary embolism

**Predisposing factors** - trauma to the extremities, hypercoagulable states (Trousseaus's syndrome in patients with carcinomas; protein C or S deficiency(as in leiden mutation); use of oral contraceptives), heart failure, pregnancy, older age, prolonged bed rest.

**Causes** - Deep vein thrombosis(most common)

**50.**   

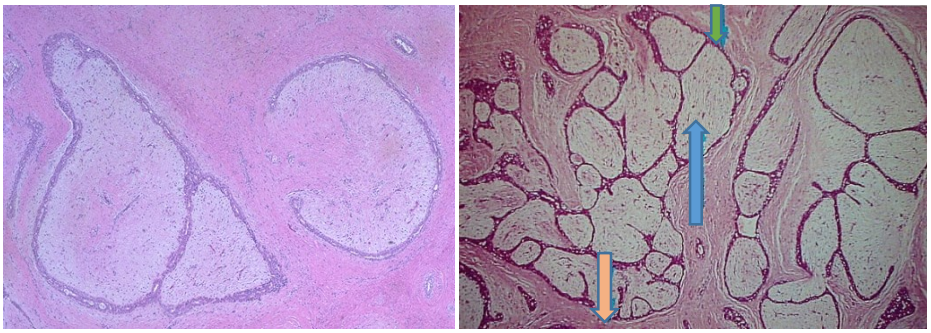
**Describe the lesion –** left slide shows a large area of infarction produced by a medium-sized thromboembolus to the lung, middle slide shows a viable alveolar architecture on the left and an area of necrosis on the right side, right slide shows the ghost architecture of an area of necrosis in a lung tissue.

**Diagnosis** – Pulmonary infarction

**What type of infarction is seen here** – Red infarct (commonly seen in loose tissues, and in tissues with dual blood supply)

**Factors that affect the development of an infarct** – Anatomy of vascular supply, rate of occlusion, tissue vulnerability to hypoxia, hypoxaemia.

**51.**



**Describe the lesion** – Slide shows compressed ducts surrounded by a fibrous stroma.(Left); The proliferation of intralobular stroma surrounds, pushes, and distorts the associated epithelium. The border is sharply delimited from the surrounding tissue.

**Gross description**: they are usually small (usually 2-4cm), solitary, sharply circumscribed freely movable nodule with smooth rounded border. The cut surface is usually firm, greyish-white, slightly myxoid and may show slit-like spaces formed by compressed ducts.

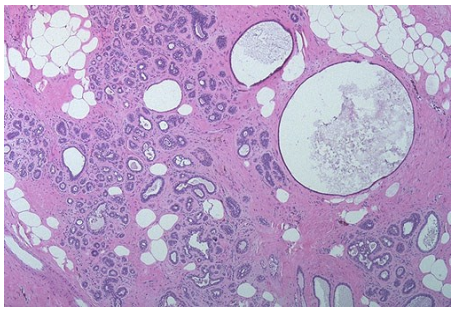
**Diagnosis** – Fibroadenoma.

**Green arrow**- epithelium

**Blue arrow**- patent glandular space

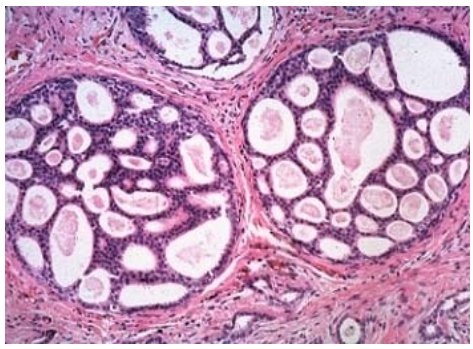
**Orange arrow**- surrounding stroma

**52.**



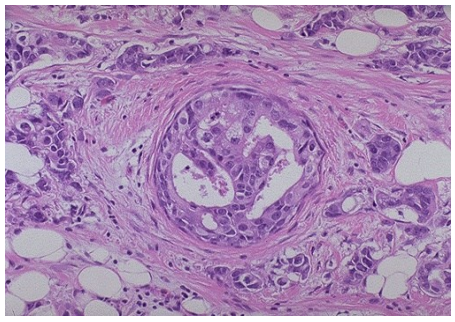
**Describe the lesion** – Slide shows the microscopic section of breast tissue with cystic dilatation of the lobules

**Diagnosis** - Fibrocystic disease of the breast

**53.** 

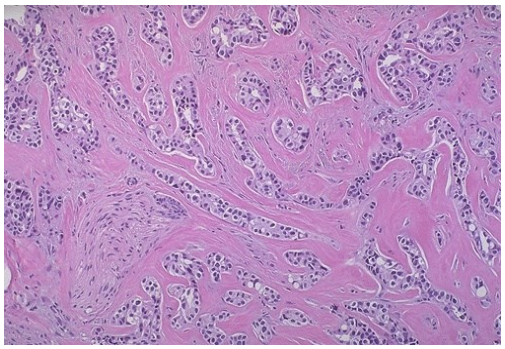
**Describe the lesion** – Slide demonstrates the cribiform appearance of a breast tumor with neoplastic cells still within the ductules. There are two large lobules at the centre of the slide with microcalcifications

**Diagnosis** - Intraductal carcinoma of the breast

**54.** 

**Describe the lesion** – Slide shows breast tissue with a ductule at the centre containing neoplastic cells infiltrating the stroma and fat cells

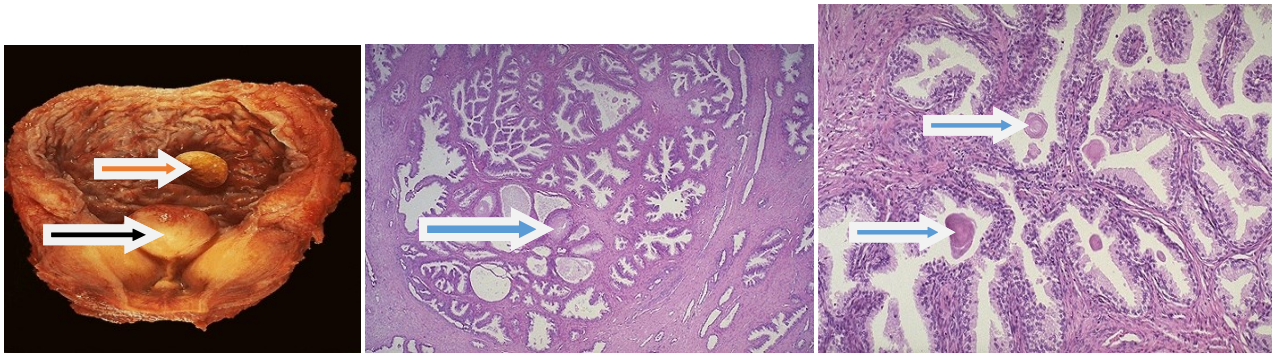
**Diagnosis** – Infiltrating ductal carcinoma of the breast

**55.** 

**Describe the lesion** – Slide shows breast tissue with infiltrating strand of neoplastic cells with bands of collagen between them. There is a nerve cell surrounded by neoplastic cells at the lower left corner of the slide

**Diagnosis** – Breast cancer

**Risk factors** – Germline mutation, First degree relatives, Race, Age of first live birth, radiation exposure, obesity, diet etc.

**56.** 

**Describe the lesion:** Macroscopically-Slide shows an enlarged prostate gland with marked increase in the size of the lateral lobes. Also seen is the prominent trabeculation of the bladder seen from the mucosal surface and the presence of yellow-brown calculi.

**Microscopically**- **(lower magnification)** slide shows glandular and stromal proliferation with the glandular proliferation being more and large hyperplastic nodules are seen as well.

**(higher magnification)** slide shows glands that well differentiated, lined by layers of epithelial cells but when nodular, a fibrous tissue surrounds the core and they still having some intervening stroma.

**Diagnosis**- Benign Prostatic hyperplasia

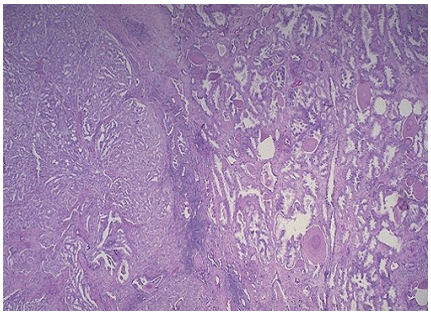
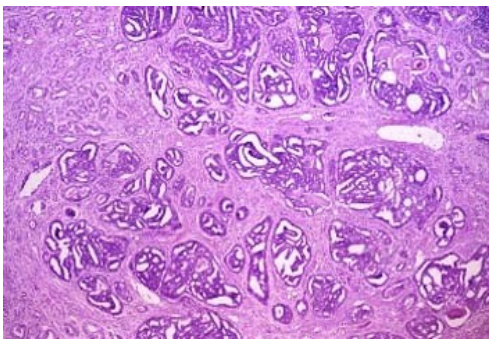
**Black arrow**- prostate

**Red arrow**-Yellow brown calculi

**Blue arrow**- corpora amylacea

**Clinical signs and Symptoms**: acute retention, dribbling (post micturition), poor streaming, increase frequency, hematuria, nocturia, hesitancy, straining, incomplete voiding.

**N/B;** Corpora amylacea is associated with old age, it is also seen in neuroglia and pulmonary alveoli in old age. They are derived from degenerated cells or thickened secretions.

**57.**  

**Describe the lesion**- **(First slide)**-Slide shows section of proliferating malignant epithelial cells forming small acinar lined back to back. The lumen becomes compressed and are hard to recognize.

**(Second slide)**- Shows higher magnification of section from prostate with malignant cell

**Diagnosis**- CA prostate

**Variants**- Prostatic Adenocarcinoma

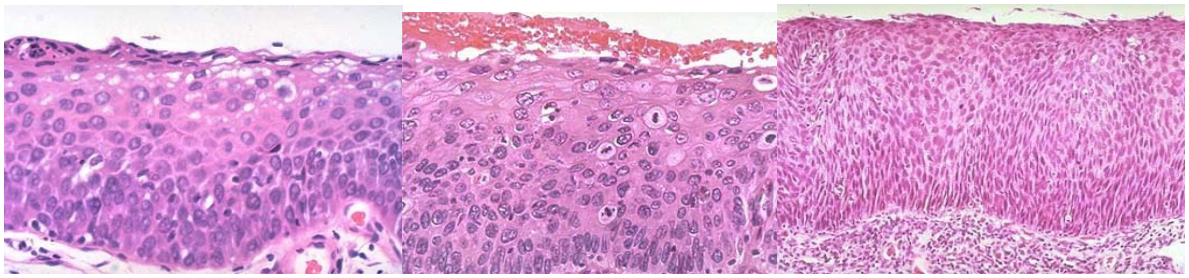
- Signet ring cell type
- Neuroendocrine cell type
- Mucinous Adenocarcinoma
- Pseudohyperplastic type

**Histological grading** –The Gleason's grading system uses a score of 1-5 for the degree of primary and secondary growth with undifferentiated and destructive types having higher scores

**Risk factors**- Black race

- Increasing age(ave. age at diagnosis is 70yrs)
- Obesity
- Family history
- Repeated untreated UTI(Gonorrhea)

**N/B:** may arise on the background of BPH and lesion is osteoblastic and usually metastasize to backbone, liver, lungs but rarely to the brain.

**58.** 

**Describe the lesion**- slides showing different stages of preneoplastic lesions. The cervical epithelium becomes progressively dysplastic which if left untreated, progresses to CA cervix.

**(First slide)**-shows preservation of epithelial maturation with koilocytes and proliferation of dysplastic cells being confined to the lower 1/3 of the epithelium with the basement membrane still intact.

**(Second slide)**-shows increasing population of dysplastic cells to about 2/3 of the epithelium. Presence of increase abnormal mitotic figures and numerous hyperchromatic and clumped nucleus.

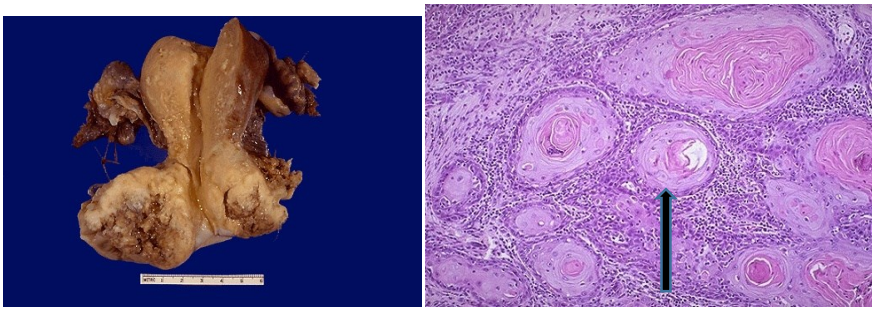
**(Third slide)**-shows full thickness involvement (severe dysplasia) i.e. Carcinoma insitu because basement membrane is still intact.

**Diagnosis**- Cervical Intraepithelial Neoplasia 1(Slide 1), CIN2(Slide 2) and CIN3(Slide 3).

**N/B:** CIN 1 and 2 are reversible.

**Risk factors**- HPV 6 and 11 infections for CIN 1 and 2.

HPV 16, 18, 30, 31 and 33 for CIN3.

**60.** 

**Describe the lesion-(Gross)**shows grayish white mass with areas of hemorrhage and necrosis within the cervix and also with spread to the vagina.

**(Histology)**-slide shows a higher magnification of nest of neoplastic squamous cells invaded through a chronically inflamed stroma. The cells are well differentiated with the presence of keratin pearls.

**Diagnosis**- CA Cervix

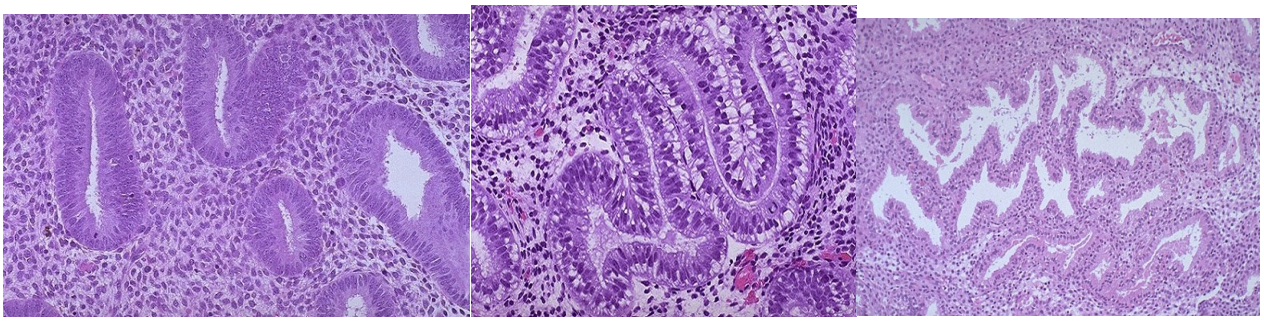
**Black Arrow**: -keratin pearls

**Variants;** Squamous cell (75%), Adenocarcinoma (15%), Adenosquamous (20%), Small cell neuroendocrine(?).

**Risk factors**-HPV infection, early age and first intercourse, multiple sexual partners, cigarette smoking, immunosuppression, multiparity, use of OCPs, certain HLA, use of Nicotine, history of mother taking diethylstilbestrol.

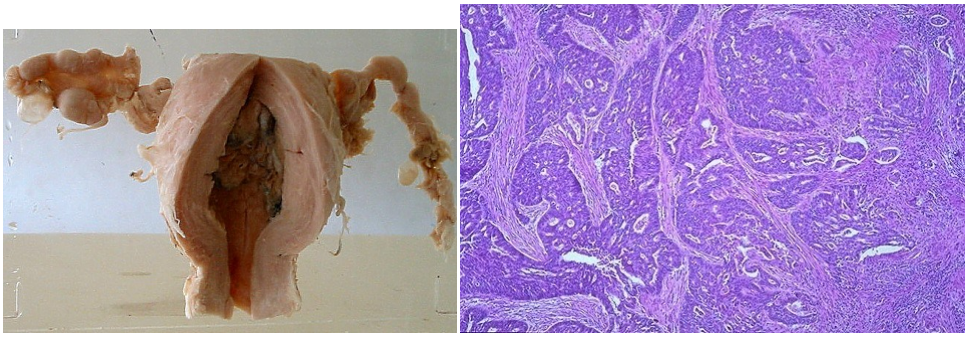
**Diagnostic Test**-Pap smear.

**62.**



**Describe the lesion- (First slide)**- Proliferative endometrium)- slide shows normal proliferative endometrial glands during the menstrual cycle. Tubular glands are seen with columnar cells with a surrounding dense stroma proliferating to produce a round pattern (doughnut appearance) with no visible blood vessels.
(**Second Slide**-Early secretory endometrium)-shows tubular glands with subnuclear vacuolations. This is consistent with post ovulatory day 2. Glands are seen to clustering to produce a saw tooth appearance.
(**Third slide**- Mid secretory endometrium)-shows tubular glands becoming more tortuous with a prominent stromal edema and also subnuclear vacuolations.

**64.**



**Describe the lesion(Gross)**-Shows an enlarged uterus with endometrial hyperplasia. There is a focus of grayish white mass with areas of hemorrhage within the lumen of the endometrium.

**(Histology)**-show a slide of bizarre looking malignant cells seen to be invading the smooth muscles of the myometrial wall of the uterus. The neoplasm has a higher stage than a neoplasm that is just confined to the endometrium or is superficially invasive.

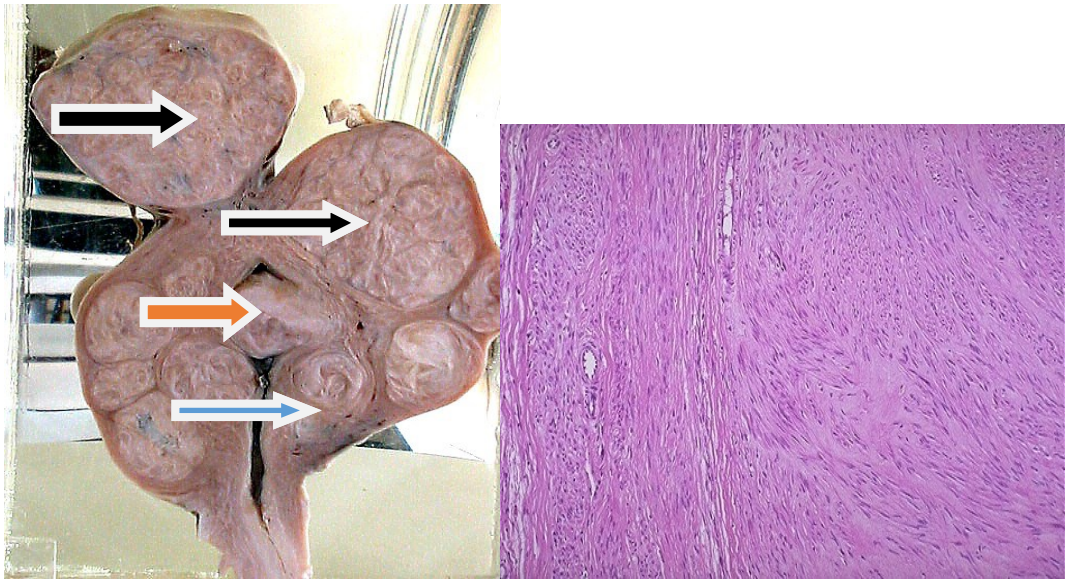
**Diagnosis-** CA Endometrium

**Variants**-Endometriod (80%), Serous.

**Risk factors**- nulliparity, unopposed estrogen secretion, ovarian tumor secreting estrogen e.g. Granulosa cell tumor, older age, HNPCC gene inheritance, Hypertension, Diabetes Mellitus, Obesity.

**Clinical presentation**-vaginal bleeding after menopause, metastasis, pelvic pain, bleeding between period, abnormal vaginal discharge, intracoital pain.

**67.**



**Describe the lesion(Gross)**-slide shows multiple greyish white, whorlly masses on different locations on the uterus. Masses are firm, round and sharply circumscribed.

(**Histology)**-slide shows partly normal(left) and neoplastic (right side) myometrium. The neoplastic cells are well differentiated and appearing in fascicles like that of the normal myometrium but are interlaced. The cells have central nuclei.

**Diagnosis-** Uterine Leiomyoma

**Arrows:** black-subserosal, blue-intramural, orange-submucosal.

**Malignant potential**-very very little or none.

**Variants**- Adenomyosis, endometriosis, benign metastasizing leiomyoma, disseminated peritoneal leiomyoma.

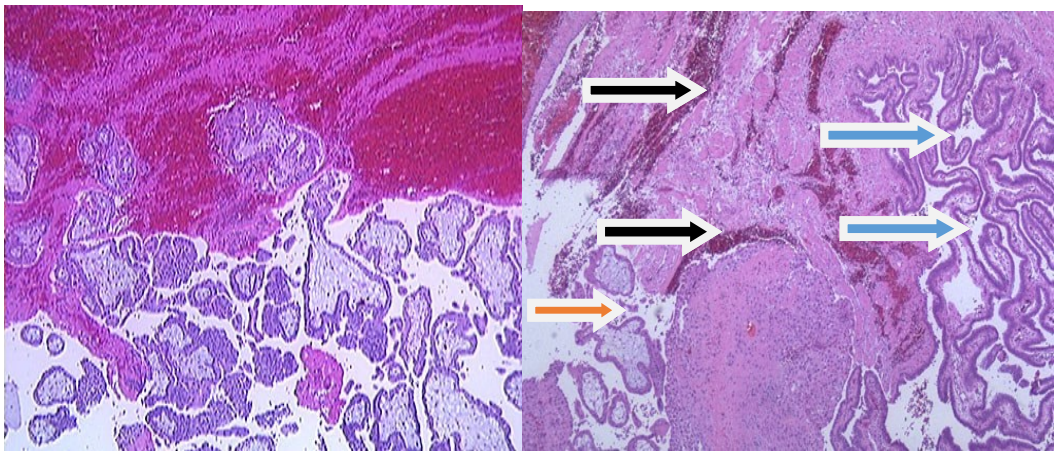
**Risk factors**-Nulliparity, obesity, positive family history, OCP use, younger females (child bearing age).

**Clinical Presentation**-Infertility, pain, constipation, abdominal destruction, bladder irritation, menorrhagia.

**Degenerative Changes**-Hyaline degeneration, red degeneration, cystic degeneration, fatty degeneration, sarcomatous change, calcification.

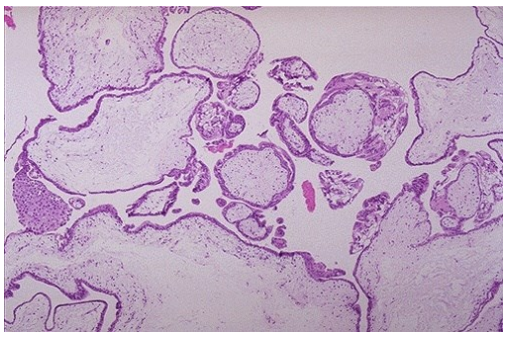
**Aetiology**- unknown

**68.**



**Describe the lesion**- (**first slide)** slide shows hemorrhagic foci and the presence of chorionic villi. Villi having central core of mesenchyme and are lined by trophoblast. Occurs following rupture of an ectopic pregnancy.
**(second slide)** Shows tubal epithelium as evident by labyrinthine folds, also present is chorionic villi tissue within the fallopian tube with areas of hemorrhage seen too.
**Diagnosis**-Products of conception/Ectopic pregnancy
Sites where these can be found-fallopian tube, ovary, abdominal peritoneu**m**
**Arrows**: **black**-engorged vessels, **blue**-labyrinthine tissues (fallopian tube tissues), **red**-chorionic villi tissue.
**Risk factors**-previous PID, previous tubal surgery, previous ectopic pregnancy.

**70.**



**Describe the lesion-(Gross)** shows grape-like cystic masses. This masses are villi which occurs as a result of trophoblastic anomalies.

**Diagnosis**- Hydatidiform mole

**Microscopic**-shows large avascular villi and areas of trophoblastic proliferations.

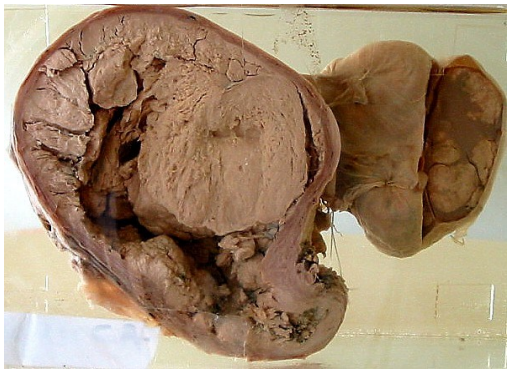
**Signs and Symptoms**-elevated HCG levels, Hypertension in second trimester, abdominal distension with no fetus, vaginal bleeding, hyper emesis gravidarum in patients, +/- bleeding, passing out grape-like villi, bilaterally enlarged cystic ovary.

**Variants**-1. Complete: Genotype; 46XX or 46XY with no fetal material and mostly entirely derived from the father. 2. Incomplete: Genotype; 69XXY with two sperms fertilizing an ovum and fetal material are usually present, commonest in developing countries.

**Risk factors**- Nulliparity, poverty, female intolerance to male tissues i.e. Spermatozoa

**N/B:** Complete moles CA.

**71.**



**Describe the lesion** – left slide shows bulky tumor-like mass occupying the uterine cavity with areas of necrosis and hemorrhage, with the ovary enlarged because of lutein cysts. Right slide tumor mass of variegated appearance occupying the uterine cavity; two nodular deposits are seen in the brain and similar nodular deposits seen in the lung.

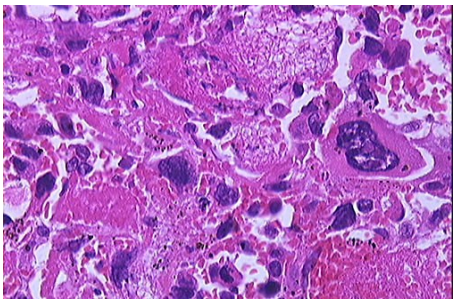
**Diagnosis-** choriocarcinoma

**Incidence**- 50% follow previous complete hydatiform mole, 25% from previous abortion, 22% from previous normal pregnancy, others from ectopic pregnancy.

Common sites of metastasis- lungs (50%) and vagina (30% to 40%), followed by, in descending order of frequency, the brain, liver, bone and kidney.

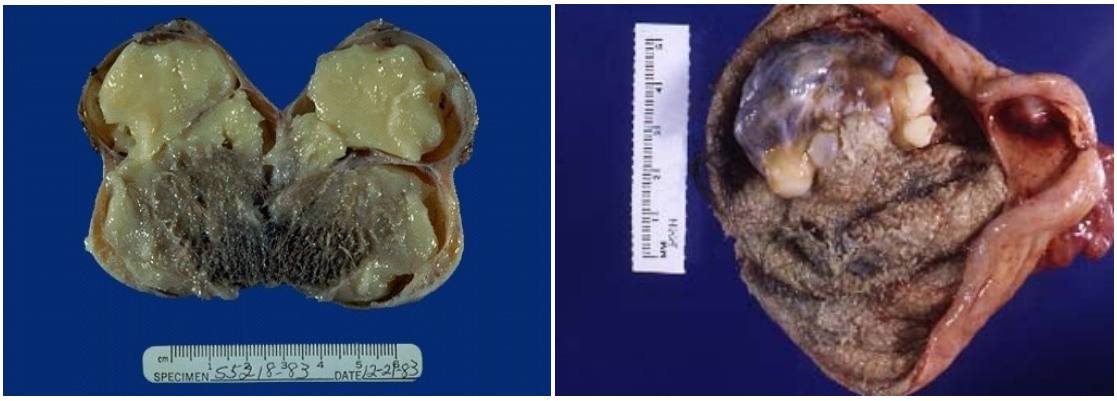
**Most common symptom-** vaginal bleeding

**Differential diagnosis-** epithelial trophoblastic tumor, placental site trophoblastic tumor, invasive Hydatidiform mole.

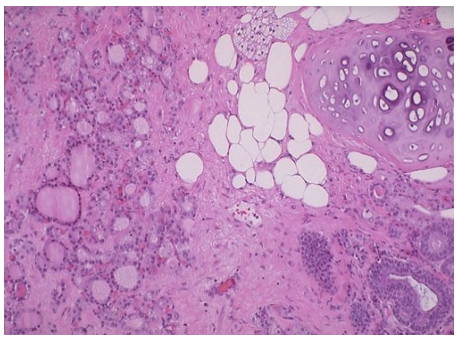
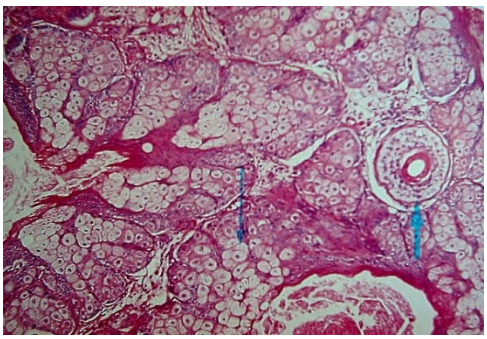
**72.** 

**Describe the lesion** – proliferation of trophoblastic cells( syncytiotrphoblast and cytotrophoblast) with hyperchromatic nuclei ; pleomorphism is well marked with areas od necrosis and hemorrhage. There is absence of chorionic villi.

**Diagnosis**- choriocarcinoma

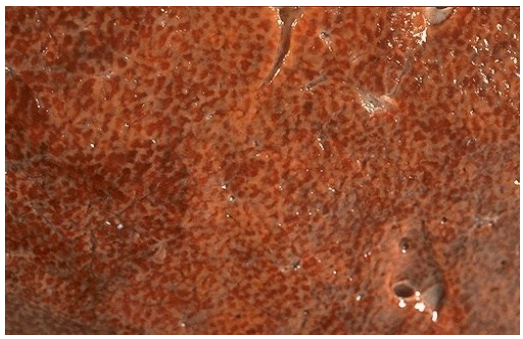
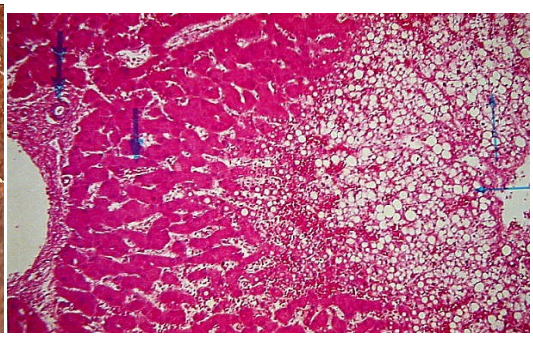
**73.** 

**Describe the lesion** – left slide shows section of the ovary with multiple cysts and strands of hair on the lower part and it containd gelationous material. Right slide large cyst with solid tumors containing teeth and strands of hair. **Diagnosis –** Dermoid cyst ovary/Teratoma

**74.**  

**Describe the lesion** – left slide shows histological section of the ovary showing cartilagenous tissue, adipocytes, sebaceous gland and thyroid gland tissue. Right slide shows cut section of the ovary showing sebaceous glands.

**Diagnosis**- benign mature teratoma of the ovary/dermoid cyst.

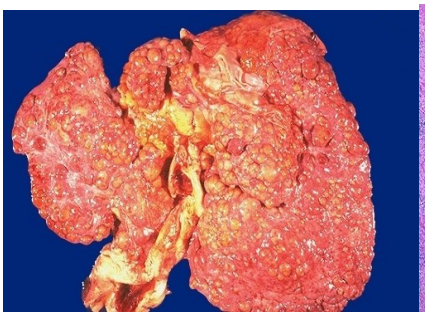
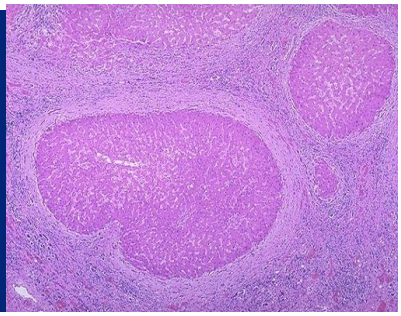
**75.**  

**Describe the lesion** –: Macroscopy: Picture shows the mottled surface of the liver with areas of yellowish discolouration alternating with dark-red areas of hemorrhagic necrosis, giving a characteristic nutmeg appearance.

**Microscopy**: Histological slide shows section of the liver tissue with an area of centrilobular necrosis, absence of distinct zone 3 hepatocytes and fatty change in other areas.

**Diagnosis**: Chronic venous congestion of the liver

**Causes**: Right heart failure, cirrhosis, sickle cell disease, hepatic vein thrombosis, DIC, intrasinusoidal metastatic tumours, peliosis hepatitis, etc.

**76.**  

**Describe the lesion** – Macroscopy: Picture shows the surface of the liver with widespread nodular distortion of its architecture, and a significant reduction in overall size

**Microscopy**: Histological slide shows section of liver tissue with pre-existing and regenerating hepatocytes surrounded by fibrous tissue; scattered lymphocytes and proliferating bile ducts are seen in areas within the fibrous septa.

**Diagnosis**: Macronodular cirrhosis

**Causes of cirrhosis**: Chronic viral hepatitis B infection (commonest in this area), excessive alcoholism, chronic viral hepatitis C infection, genetic/metabolic diseases (hemochromatosis, cystic fibrosis, alpha-1 antitrypsin deficiency, Wilson's disease), autoimmune hepatitis, primary biliary cirrhosis etc.

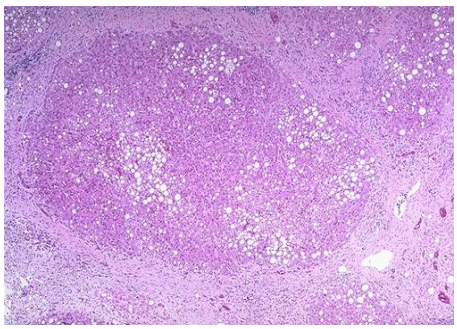
**Nodules**- Macronodules: >3mm in diameter. Commonly caused by chronic hep. B infection

Micronodular: <3mm in diameter. Commonly caused by alcoholism

Mixed: Macronodular + micronodular

**Clinical features**: Some individuals with cirrhosis are asymptomatic; other features are anorexia, weight loss, weakness, jaundice, peripheral edema, irrational behaviour, irregular sleep patterns, hepatorenal syndrome, hepatopulmonary syndrome

**Complications**: Portal hypertension, splenomegaly, portosystemic encephalopathy, steatorrhea, hepatocellular carcinoma

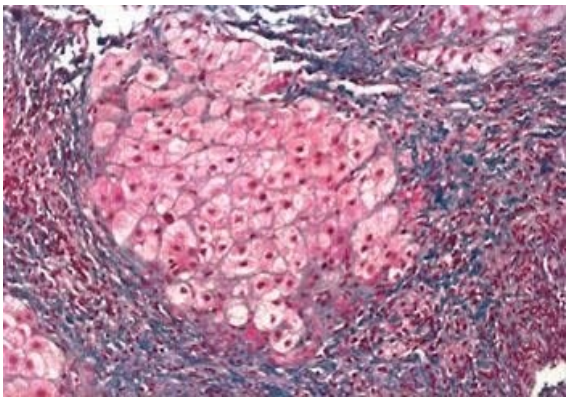
**77.**  

**Describe the lesion** – Macroscopy: Similar to macronodular cirrhosis

**Microscopy**: Histological slide of the liver tissue showing areas of demarcation of hepatocytes by fibrous septa with moderate fatty change.

**Diagnosis**: Micronodular cirrhosis

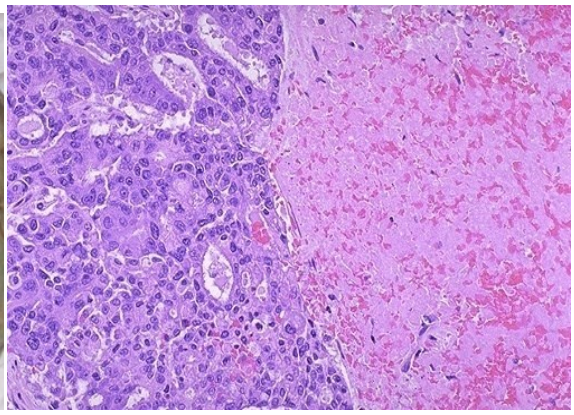
**Causes, clinical features and complications**- refer to previous number

**78.** 

**Describe the lesion**: That of liver cirrhosis

**Diagnosis**: Liver cirrhosis

**Special stain**: Masson trichome stain. It gives the fibrous tissue around the hepatocytes a blue or green colour, depending on the counterstain used, which could be methylene blue or malachite green

**79.**  

**Describe the lesion** - **Macroscopy**: Picture shows the surface of the liver with a greyish-white irregularly

shaped mass, areas of haemorrhage and necrosis. Areas of nodularity are also seen.

**Microscopy**: Histological slide shows section of liver tissue with areas of necrosis and hemorrhage on the right

side, and proliferation of malignant hepatocytes having hyperchromatic nuclei on the left.

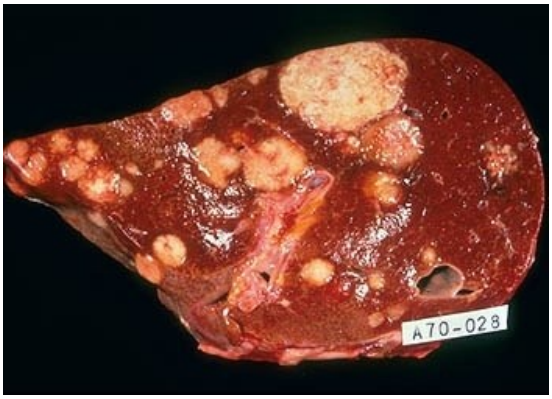
**Diagnosis**: Primary liver cell carcinoma

**Predisposing factors**: Chronic HBV or HCV infection, chronic alcoholism, non-alcoholic steatohepatitis, aflatoxin- contaminated food, alpha-1-antitrypsin deficiency, thorotrast administration, *Clonorchis sinensis* infection etc.

PLCC arises from any component of the liver; HCC arises from the hepatocytes.

**Tumour marker for HCC**- alpha-fetoprotein

**80.**



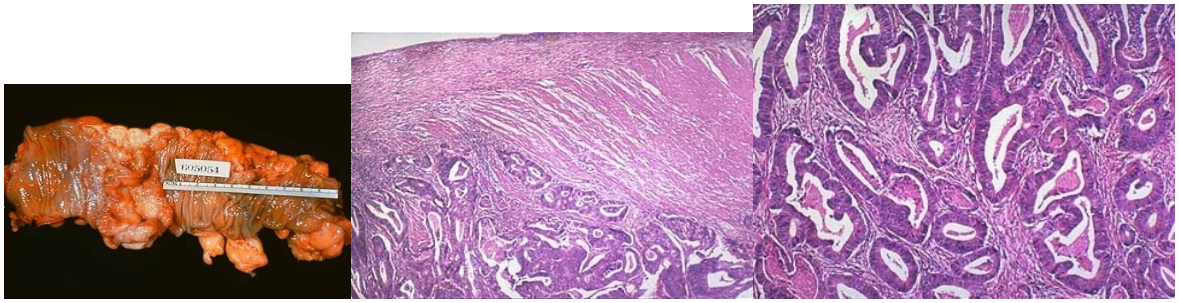

**Describe the lesion**: Picture shows the surface of the liver appearing hyperaemic, enlarged and oedematous, with areas of greyish-white discolouration having central umbilication.

**Diagnosis**: Metastatic adenocarcinoma to the liver.

**Predisposing factors**: Malignancies from other sites e.g. colon, breast, lung, pancreas, or any cancer in any site of the body.

**Important features to note on gross examination.**

| **PLCC** | **Metastatic liver disease** |
|---|---|
| 1) No central umbilication is seen | Central umbilication is seen on the lesion |
| 2) Here, there is usually a background of cirrhosis, so nodules are visible | No nodules are seen |

**81.** 

**Describe the lesion** – Macroscopy: Picture shows the colon with a greyish-white bulky cauliflower-shaped mass interrupting the normal structure; the colon appears hyperaemic and haemorrhagic.

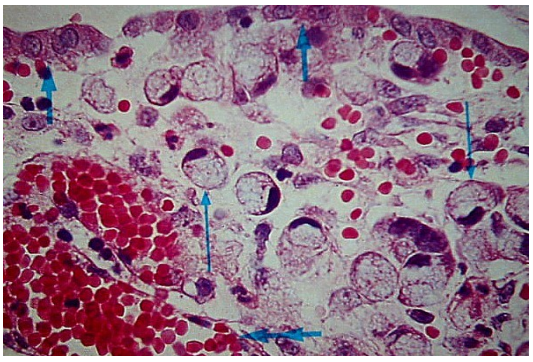
**Microscopy**: Histological slide shows section of the colonic tissue with proliferation of the glandular components having hyperchromatic nuclei invading the muscular layer. Glands are seen to contain mucin, and are irregularly shaped.

**Diagnosis**: Adenocarcinoma of the colon

**Predisposing factors**: Age, diet (low fibre, high fat, low consumption of micronutrients), obesity, familial polyps, inflammatory bowel disease (commonly ulcerative colitis)

**Symptoms/ presenting complaints**- Change in bowel habit, haematochezia, vomiting, weight loss etc. Obstructive features are common with left-sided malignancies; haemorrhagic features are common with right-sided malignancies

**Gene mutation pathways for colorectal carcinoma**- APC/ β-catenin pathway, microsatellite instability pathway.

**82.** 

**Describe the lesion** – Histological slide shows malignant epithelial cells containing abundant intracellular mucin, pushing the nuclei to the periphery giving a signet-ring appearance. Distended blood vessels are also seen.

**Diagnosis**: Mucinous adenocarcinoma (Krukenberg tumour)

**Krukenberg tumours**- bilateral metastases of gastric, pancreatic and colorectal mucinous adenocarcinomas to the ovaries. Mucinous adenocarcinomas arising from surface epithelium of the ovary are commonly unilateral.

**83.** 

**Describe the lesion** – Picture shows the surface of the ileum with presence of raised Peyer's patches and multiple round to oval areas of ulceration and necrosis lying longitudinally.

**Diagnosis**: Typhoid enteritis/ Typhoid ileitis

**Causative organism**: *Salmonella typhi*

**Route of infection**: Feco-oral

**Samples useful for laboratory diagnosis**: Blood, stool and urine.

*Blood samples are positive in the first week of infection, stool samples are positive in the second week, urine samples are positive in the third week.*

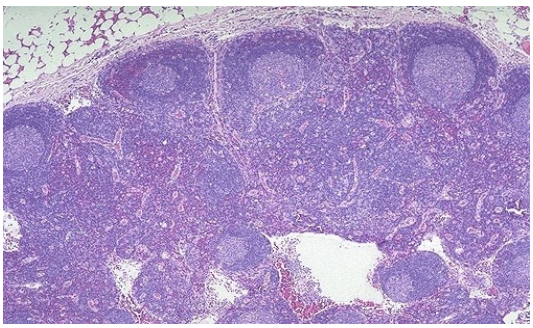
Others less commonly used include bile and synovial fluid aspirate.

**Clinical features**: High fever, headache, weakness, GI symptoms, including abdominal pains and diarrhea or constipation; loss of appetite, rash of flat, rose-colored spots, Faget's sign

**Complications**: Perforation, lower GI bleeding, cholecystitis, typhoid myocarditis, typhoid pericarditis, typhoid meningitis, typhoid psychosis, typhoid pneumonitis, typhoid osteomyelitis (common among sickle cell disease patients), Zenker degeneration, etc.

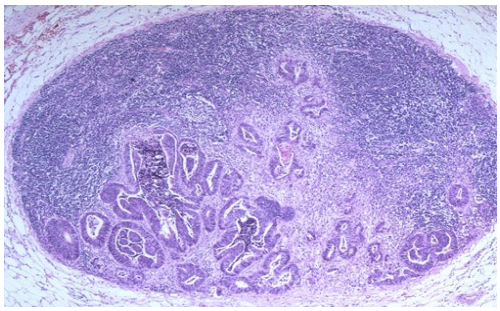
NB: TB ulcers in the intestine lie transversely, typhoid ulcers lie longitudinally, amoebic ulcers are flask-shaped.

TB ulcers in the intestine heal by fibrosis, typhoid ulcers do not.

**84.** 

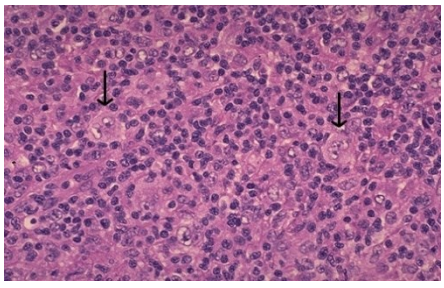
**Describe the lesion** – Histological slide shows section of a lymph node with pale staining germinal centres, and proliferation of the lymphoid cells in the cortex and paracortex. The capsule and subcapsular sinuses are also seen.

**Diagnosis**: Reactive lymph node Causes: infections e.g. HIV and other viral infections, toxoplasmosis, etc.,

**85.** 

**Describe the lesion** – Histological slide of a lymph node showing proliferation of malignant glandular cells, with reactive hyperplasia of the lymphoid cells and enlargement of the lymph node.

**Diagnosis**: Metastatic adenocarcinoma of the lymph node

**86.** 

**Describe the lesion –:** Histological slide shows numerous reactive lymphoid cells, separated in areas by the presence of large cells with large, pale nuclei containing purple-staining nucleoli, giving them a characteristic owl-eye appearance.

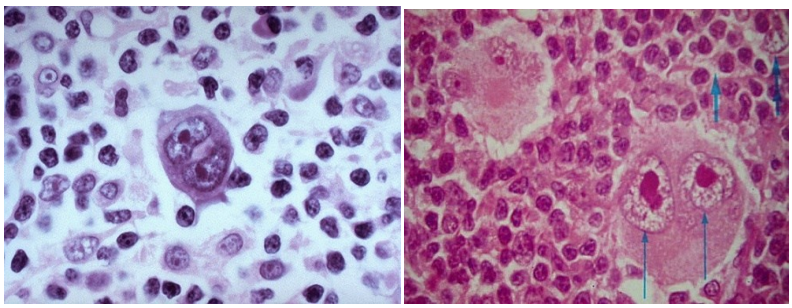
**Diagnosis**: Hodgkin's lymphoma

**Characteristic feature**: Reed-Sternberg giant cells

**Variants**: Classical HL (Nodular sclerosis, mixed cellularity, lymphocyte rich, lymphocyte depleted), Nodular lymphocyte predominant HL

Most common in the developed countries: NS; most common in underdeveloped countries: MC, LD; Rarest: LD.

**Clinical features**: Painless lymphadenopathy, fever, night sweats, weight loss.

**87.** 

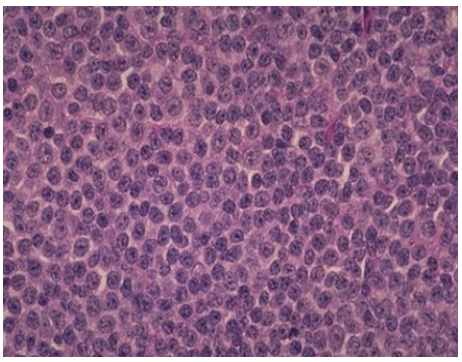
**Describe the lesion** – Histological slide showing a large cell with two nuclei or a bilobed nucleus, each measuring about 12-18mm in diameter; each nucleus has an eosinophilic nucleolus, with a clear zone around it, giving a characteristic “owl eye” appearance.

**Diagnosis**: Reed-Sternberg giant cells in Hodgkin lymphoma

Two conditions in which giant cells resembling the RS cells (RS-like cells) are seen: infectious mononucleosis, large B cell lymphoma

**Variants of RS cells**: Classical, lacunar, mononuclear, pleomorphic, lymphohistiocytic/popcorn cell variant.

Popcorn cell variant is usually CD15-, CD30- and CD45+. Other variants are CD15+, CD30+, CD45-.

**88.** 

**Describe the lesion** – Histological slide shows the presence of a massive infiltrate of inflammatory cells having hyperchromatic nuclei and intracellular debris. Large multinucleated cells are absent.

**Diagnosis**: Non-Hodgkin lymphoma

WHO classification: tumours of B cells, tumours of T cells, Hodgkin lymphoma

Staging for HL & NHL: Ann-Arbor staging

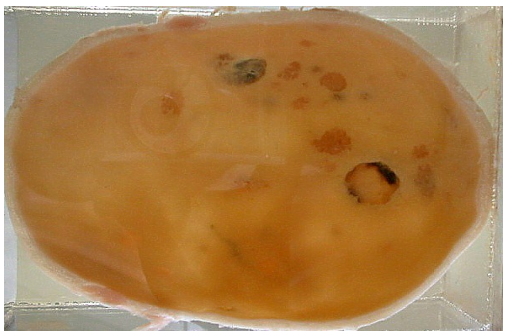
**89.** 

**Describe the lesion** – Picture shows the spleen appearing enlarged, and cut surface with a greyish-white discolouration due to accumulation of hemozoin pigments.

**Diagnosis**: Splenomegaly secondary to malarial infection.

Causes of splenomegaly include kala azar, CML, HCL, CLL, myelofibrosis, Gaucher syndrome, malaria, etc.

Splenomegaly- enlarged spleen > 250g

**90.** 

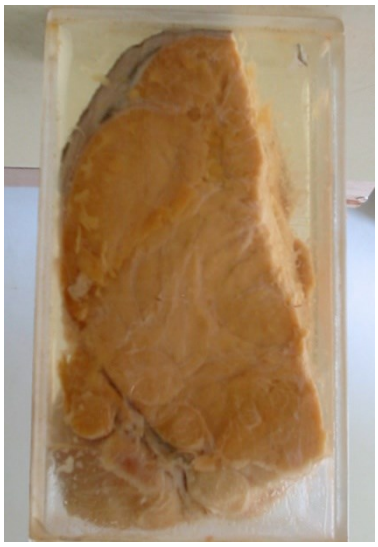
**Describe the lesion** – Picture shows the cut-surface of the skull with presence of multiple osteolytic punched-out lesions.

**Diagnosis**: Multiple myeloma

**Primary cells involved**: Plasma cells.

Paraproteins produced could be IgG, IgA, IgE, IgD, or light chains only.

**Complications**: Immunosuppression, hypercalcemia, anemia, amyloidosis, carpal tunnel syndrome, renal failure, hyperuricemia, etc.

**91.** 

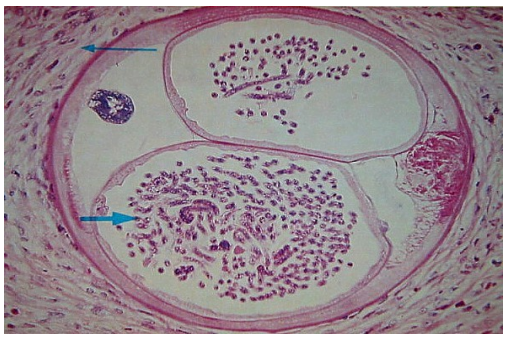
**Describe the lesion** – Picture shows a yellowish well-encapsulated mass of mature adipocytes.

**Diagnosis**: Lipoma

**Common body sites**: Proximal extremities, trunk, gluteal region, scalp.

**Mean age of occurrence**: 40-60 years

Important diagnostic criterion for malignant counterpart of this condition: Presence of malignant cells especially the lipoblast.

**92.** 

**Describe the lesion** - Histological slide shows section of a gravid female onchocerca with microfilariae surrounded by areas of fibrosis and inflammatory infiltrates.

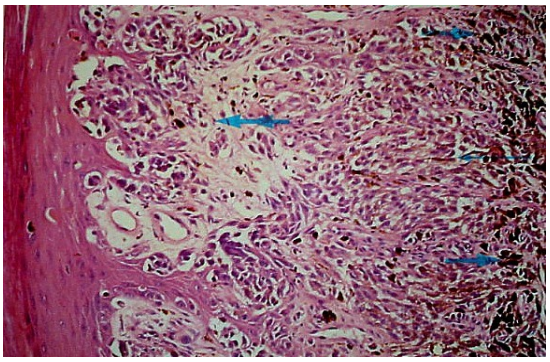
**Diagnosis**: Onchocerciasis

**Characteristic subcutaneous nodule**: Onchocercoma

**Vector***: Simulium spp* of flies (blackflies)

Characteristic response to treatment with antifilarial drugs: Mazzotti reaction

**Drug of choice**: Ivermectin

**93** 

**Describe the lesion** - Histological slide shows section of the skin and stratified squamous epithelium with infiltration of the stroma by proliferating melanocytes containing black-brown pigment.

**Diagnosis**: Malignant melanoma

**Other commonly affected sites**: oesophagus, oral mucosa, anogenital mucosa., retina, meninges

**Measure of metastatic potential**: Breslow thickness

# about us

## VISION

That God made man - spirit, soul and body, and He has called us Christian in the medical and dental disciplines to be proficient in caring for the whole man (1 Thess 5:23).
Hence, the Christian Medical and Dental Association of Nigeria (CMDA Nigeria) seeks to establish a Christian witness through medical and dental doctors and students in every community in Nigeria and
beyond.

## MISSION

**1**. To start and strengthen Christian Medical and Dental Association of Nigeria (CMDA Nigeria) chapters nationally through calling, equipping, fellowship and service.
**2**. To establish and enhance Christian witness through members' personal and corporate ministry in holistic care and in mission outreaches.

## PASSION

Our mission is sustained by our passion as future doctors to be different; our commitment to seeing patients as real human beings and not just mere cases. Our burning desire to be used by God to restore the dignity to men and also to reconcile them to their creator in the steps of the great physician, our Lord Jesus.

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